RESIDENTIAL RADON CONTAMINATION AND INDOOR QUALITY RESEARCH NEEDS

HEARING

BEFORE THE

SUBCOMMITTEE ON NATURAL RESOURCES,
AGRICULTURE RESEARCH AND ENVIRONMENT

OF THE

COMMITTEE ON
SCIENCE AND TECHNOLOGY
U.S. HOUSE OF REPRESENTATIVES

NINETY-NINTH CONGRESS

SECOND SESSION

SEPTEMBER 17, 1986

[No. 159]

Printed for the use of the
Committee on Science and Technology

U.S. GOVERNMENT PRINTING OFFICE
65-340 O WASHINGTON : 1987

For sale by the Superintendent of Documents, Congressional Sales Office
U.S. Government Printing Office, Washington, DC 20402

H701-40

COMMITTEE ON SCIENCE AND TECHNOLOGY

DON FUQUA, Florida, *Chairman*

ROBERT A. ROE, New Jersey
GEORGE E. BROWN, Jr., California
JAMES H. SCHEUER, New York
MARILYN LLOYD, Tennessee
TIMOTHY E. WIRTH, Colorado
DOUG WALGREN, Pennsylvania
DAN GLICKMAN, Kansas
ROBERT A. YOUNG, Missouri
HAROLD L. VOLKMER, Missouri
BILL NELSON, Florida
STAN LUNDINE, New York
RALPH M. HALL, Texas
DAVE McCURDY, Oklahoma
NORMAN Y. MINETA, California
BUDDY MacKAY, Florida **
TIM VALENTINE, North Carolina
HARRY M. REID, Nevada
ROBERT G. TORRICELLI, New Jersey
RICK BOUCHER, Virginia
TERRY BRUCE, Illinois
RICHARD ~~H. STALLINGS~~, Idaho
BART GORDON, Tennessee
JAMES A. TRAFICANT, Jr., Ohio
JIM CHAPMAN, Texas

MANUEL LUJAN, Jr., New Mexico *
ROBERT S. WALKER, Pennsylvania
F. JAMES SENSENBRENNER, Jr., Wisconsin
CLAUDINE SCHNEIDER, Rhode Island
SHERWOOD L. BOEHLERT, New York
TOM LEWIS, Florida
DON RITTER, Pennsylvania
SID W. MORRISON, Washington
RON PACKARD, California
JAN MEYERS, Kansas
ROBERT C. SMITH, New Hampshire
PAUL B. HENRY, Michigan
HARRIS W. FAWELL, Illinois
WILLIAM W. COBEY, Jr., North Carolina
JOE BARTON, Texas
D. FRENCH SLAUGHTER, Jr., Virginia
DAVID S. MONSON, Utah

Harold P. Hanson, *Executive Director*
Robert C. Ketcham, *General Counsel*
Regina A. Davis, *Chief Clerk*
R. Thomas Weimer, *Republican Staff Director*

Subcommittee on Natural Resources, Agriculture Research and Environment

JAMES H. SCHEUER, New York, *Chairman*

TIMOTHY E. WIRTH, Colorado
TIM VALENTINE, North Carolina
DAVE McCURDY, Oklahoma
HARRY M. REID, Nevada
ROBERT G. TORRICELLI, New Jersey
MICHAEL A. ANDREWS, Texas

CLAUDINE SCHNEIDER, Rhode Island
DON RITTER, Pennsylvania
JAN MEYERS, Kansas
ROBERT C. SMITH, New Hampshire
PAUL B. HENRY, Michigan

---

* Ranking Republican Member.

** Serving on Committee on the Budget for 99th Congress.

CONTENTS

WITNESSES

September 17, 1986: Page
- Donald Ehreth, Acting Assistant Administrator, Office of Research and Development, Environmental Protection Agency, accompanied by Sheldon Myers, Deputy Director, Office of Radiation Programs; and Charles de Lisi, Associate Director, Office of Health and Environmental Research, Department of Energy, accompanied by John P. Millhone, Director, Office of Buildings and Community Systems, Conservation and Renewable Energy 4
- Dr. Jan Stolwijk, Chairman, Department of Epidemiology and Public Health, Yale University School of Medicine; Richard Zimmer, Chairman, National Council for Clean Indoor Air, accompanied by Ken Sexton, Director of Research, Health Effects Institute, and A. Judson Wells, Vice Chairman, National Council for Clean Indoor Air 32

APPENDIX

Press release from Hon. Claudine Schneider 101
Material submitted for the Record by Donald J. Ehreth, Acting Assistant Administrator for Research and Development, U.S. Environmental Protection Agency 1986. Keynote address at the Air Pollution Control Association Conference, June 23, 1986. Letter from Harvard University, School of Public Health, with the findings of the Ad Hoc Review Panel "An Agenda for Research: Indoor Environments." Copy of EPA Journal, volume 12, number 6, August 1986 on "Radon: Pinpointing a Mystery." 103
Letter from A. Judson Wells with additional answers to questions 179
Statement for the Record from the Tobacco Institute 249
Statement for the Record from the Commonwealth of Pennsylvania, department of environmental resources 265
Statement for the Record from the Radon Information Council 269
Statement for the Record from Gray Robertson, ACVA Atlantic Inc 277
EPA Response to questions submitted for the record 307

RESIDENTIAL RADON CONTAMINATION AND INDOOR QUALITY RESEARCH NEEDS

WEDNESDAY, SEPTEMBER 17, 1986

House of Representatives,
Committee on Science and Technology,
Subcommittee on Natural Resources,
Agriculture Research and Environment,
Washington, DC.

The subcommittee met, pursuant to call, at 1:15 p.m., in room 2318, Rayburn House Office Building, Hon. James H. Scheuer (chairman of the subcommittee) presiding.

Mr. Scheuer. Good afternoon. The Subcommittee on Natural Resources, Agriculture Research and Environment will come to order.

Today, the subcommittee returns to a topic which is of particular interest to me and to our distinguished ranking member, Claudine Schneider. And here she is, entering on cue. Let me say that she has been a driving force behind the congressional interest in radon and the progress that we have made legislatively. We wouldn't be here if she hadn't had the commitment and the energy and shown the leadership that she has. She has been a very driving force in this whole area over the past 5 years.

Since our first hearing on this subject 5 years ago, the perils of asbestos, formaldehyde, environmental tobacco smoke, and now radon, have become matters of common concern in homes, offices, and schools all across the country. But it is still hard for people to understand and accept the fact that we face greater health risks from airborne toxic chemicals indoors than we do from all of the airborne toxics outdoors. That reluctance may explain, in part, why indoor air pollution doesn't command the attention or the funding which it deserves.

Despite the fact that radon accounts for more lung cancer deaths than all outdoor air pollution put together, EPA requested only $2 million for indoor studies of radon in fiscal year 1987, only 3 percent of the amount that will be devoted to research on outdoor air pollution. It simply doesn't make sense. Even more curious is the fact that the Office of Management and Budget consistently opposes funding any research whatsoever on indoor air quality. It is mindboggling.

To change this situation, Congresswoman Schneider and I introduced a bill last year directing EPA to conduct an indoor air research program aimed at assessing health risks and developing methods to reduce those risks. I am pleased to say that major provisions of that bill have now been incorporated into the Superfund legislation now pending in the Conference Committee.

And I want to take this opportunity again of congratulating Congresswoman Schneider for her hard work and her tremendous leadership on this significant achievement.

Last October, the subcommittee held hearings on what was then a relatively unknown problem: radon in homes. As our witnesses predicted then, radon has since become a matter of national concern with discoveries of high radon levels being made in nearly every part of the country.

Despite this concern, the GAO—the Government Accounting Office—which is the investigative arm of the Congress, has recently charged that no overall Federal strategy exists to address the indoor radon problem, even though indoor radon accounts for more cancer deaths than all outdoor pollution put together.

Today, the subcommittee will examine the radon programs of the two major Federal agencies involved—the Department of Energy and the Environmental Protection Administration. While radon is quite appropriately the subject of great concern, it is only one part of the larger indoor air quality issue, despite the efforts of some who would portray it as a unique issue like asbestos, formaldehyde and cigarette smoke in the past.

But that larger issue is rapidly getting lost in the stampede. DOE has stopped funding health effects research on anything but radon, and the group charged with developing an overall comprehensive strategy, a coordinated and comprehensive indoor air strategy, the Interagency Committee on Indoor Air Quality, has scarcely agreed on anything but its name in the last 2 years.

It is time to stop pretending that indoor air quality and pollution is a problem that will go away if only we ignore it long enough. It is time we recognized that indoor air pollution, and in particular, radon, is a major public health issue that must be faced. Congress shares the responsibility for providing a new direction for Federal indoor air pollution programs, and our hearing today, we hope, will be a step in the right direction in defining such a new initiative, and we hope it will be a step in creating the momentum for achieving that new initiative.

Madam Schneider.

Miss SCHNEIDER. Yes, sir. Well, Mr. Chairman, I think that certainly your comments about the extent of the problem were very comprehensive. What I would like to add, however, is that there is growing public awareness of this problem. Mr. Scheuer and I began to address it I think before there was such public awareness, and I think we are only touching the tip of the iceberg insofar as information generated about the extent of the problem is concerned.

You may be aware that on Tuesday of this week The Washington Post did an article about indoor air pollution, for which I am very grateful, and the Consumer Federation of America has also designated indoor air pollution as the concern of the 1980's and 1990's. The environmental top No. 1 issue. I think that this is appropriate because this body all too often has the tendency to focus on crises management. And I think that those of us and those of you witnesses who are gathered here today have looked into the breadth and the depth of the problem and acknowledged that we cannot afford to wait till we have all of the information, but we must proceed expeditiously to devise a time schedule and a specific game plan as to how we are going to, No. 1, understand the degree of the problem of indoor air pollution, and, second, what are the mitigating measures that we might be taking.

As was already pointed out, the average individual spends about 75 percent of their time indoors. But of course, for the elderly, for handicapped and also for small children, that number is much higher. And having faced the energy crisis and insulated our homes to a tighter degree, we have not made provisions for the kind of ventilation that would exchange air appropriately so that we would not be exposed to the concentrations to which we are currently exposed.

And then when one takes into characterization that we have not only the typical cigar and cigarette smoke, but we also have exposure to carpet adhesives, furniture resins, cleaning materials, not to mention kerosene- and wood-heating exhaust, then we know that our potential problems are being compounded. And when 12,000 to 20,000 lung cancers of the 120,000 lung cancers that occur each year are believed to be attributable to indoor air pollution, more specifically radon, then certainly that message is loud and clear that we need some action.

It is disconcerting to me that, I believe that EPA is not developing the information that the States need in a timely manner because EPA has failed to develop a research policy statement to guide the indoor air pollution efforts and, second, EPA has spent scarce resources to plan a multipollutant field survey which will not address the most critical data gaps. So I think that it is imperative that we have at least those two criteria out of the way and move along to resolving this problem before we look at the explosion in health care costs and retrofit costs which are more likely than not about to appear.

Thank you for allowing us to have this hearing, Mr. Chairman, and I look forward to the information we will receive from it. Thank you.

Mr. SCHEUER. Very good.

Congressman Bart Gordon of Tennesee.

Mr. GORDON. Mr. Chairman, I want to thank you for the opportunity to join your subcommittee hearing this afternoon on EPA's progress in tackling the increasing problem of indoor radon contamination, the second leading cause of lung cancer. And I commend the chairman and Congresswoman Schneider for your insight and leadership in this important issue.

Middle Tennessee, like other areas of the country, is targeted as a hot spot for radon contamination. A recent radon survey of nearly 500 Tennessee homes revealed some disturbing results.

Over 27 percent of the homes recorded high level radon exposure, which is equal to 200 chest x rays per year. Scientists estimate that long-term exposure at this level increases by five times a nonsmoker's chances of having lung cancer.

An additional 5 percent of the homes recorded radon exposure levels equal to 20,000 chest x rays per year, the equivalent of smoking four packs of cigarettes a day. This increases the chances of having lung cancer over 60 times.

And although these tests indicate that almost one-third of Tennessee homes have what EPA terms as action levels of radon, no one really knows for sure to what extent this health hazard is in Tennessee or which neighborhoods, homes and schools might be contaminated.

So, Mr. Chairman, I am here today to question EPA on its efforts, both nationally and specifically in Tennessee, in identifying the possible areas of contamination, developing a plan of attack to lessen the hazard, and to prevent further occurrences. So thank you for letting me join you today.

Mr. SCHEUER. Thank you very much, Congressman.

All right, we will go ahead with the first panel, which is the Federal panel, and we will hear first from Don Ehreth, Acting Assistant Administrator of the Office of Research and Development of EPA, accompanied by Sheldon Meyers, Deputy Director of the Office of Radiation Programs. Then we will hear from Charles DeLisi, Associate Director of the Office of Health and Environmental Research of the Department of Energy, who also is accompanied by John Millhone, Director of the Office of Buildings and Community Systems, Conservation and Renewable Energy.

All right, Mr. Ehreth, I will ask you, and all the other witnesses, to take 5 minutes and sum up your remarks. Of course your printed testimony will be printed in full at the point in the record at which you speak. So please sum up as informally as you can; try not to read, just talk to us; and then I am sure we will all have some questions for you.

STATEMENTS OF DONALD EHRETH, ACTING ASSISTANT ADMINISTRATOR, OFFICE OF RESEARCH AND DEVELOPMENT, ENVIRONMENTAL PROTECTION AGENCY, ACCOMPANIED BY SHELDON MEYERS, DEPUTY DIRECTOR, OFFICE OF RADIATION PROGRAMS, AND CHARLES DeLISI, ASSOCIATE DIRECTOR, OFFICE OF HEALTH AND ENVIRONMENTAL RESEARCH, DEPARTMENT OF ENERGY, ACCOMPANIED BY JOHN P. MILLHONE, DIRECTOR, OFFICE OF BUILDINGS AND COMMUNITY SYSTEMS, CONSERVATION AND RENEWABLE ENERGY

Mr. EHRETH. Thank you, Mr. Chairman. I appreciate the opportunity to have our statement provided for the record and printed in full. I would like to do just that, just sort of summarize where we have been, where we are at now, where I think we are going; and I hope to do that in the 5 minutes.

In the past 3 to 4 months, our indoor air research program has taken a dramatic change in direction. The field survey alluded to by Miss Schneider is, in fact, something that we have set on the side burner, or back burner, if you will. As a matter of fact, I might suggest that it is probably off the stove by now. But in any case, that represents a very dramatic shift.

But how did we get there? Why did we even embark upon such a course of action?

In 1983, I sat before you—August 1983—and I came here with good news, that EPA was supporting the conduct of research in indoor air. And at that point in time I laid out about four questions that I thought we might be able to address in indoor air research.

What are indoor air pollutants of concern, their concentrations, and their sources? What are the parameters affecting their strengths inside buildings or homes? What are the relationships between indoor air pollution, building design and all the other factors associated with indoor air pollution health effects? And what is the magnitude of the problem in terms of exposure, health effects, and finally risks? And finally, and certainly by all means just as important, what are the most cost-effective ways to reduce these concentrations or risks?

We developed a plan—the fact is, we developed two plans. We took it before a scientific panel, which is normal procedure for us to do when we embark upon a new program. And we received some guidance from that peer review panel. It was basically that with the resources available it would not be prudent to undertake a broad-based, comprehensive research program, but to focus on some near-term objectives. And one of those near-term objectives was to facilitate or to develop the capability to eventually put into place a multipollutant field survey.

This, initially, was also reflected in some of the congressional language of reports and things of that nature. So we embarked upon that, and I think that, quite frankly, we accomplished quite a considerable amount that can be used in the future or now or whenever. We have developed new methods of instrumentation for monitoring air pollutants in the homes and in buildings. We have gotten a much better understanding of the role of environmental tobacco smoke, and we have recognized now that it is probably one of the most potent mutagens found indoors, as well as the mutagens—byproducts of combustion from kerosene heaters and the mutagenic property of same, is what I meant to say.

In the meantime, probably one of the most significant successes that has come out of the Indoor Air Pollution Program has been the success we have enjoyed with regard to mitigating risks associated with radon exposure, and Mr. Myers will summarize the Radon Program in just a moment.

But nevertheless, after August 1983, I began to come back here as the Deputy Assistant Administrator and we began to speak in terms of not being able to support or carry out a significant enough of a discussion or a proposal for a sustained priority for the indoor air program. Consequently, as you identified earlier, in 1985 and 1986, we did not have those resources in our program. We recognize that.

But that in itself led to some of the reasons for why we funded things as we did. We operated on a year-to-year basis. We funded projects that we felt could be completed within those resources available, and so consequently we were looking for short-term gains but positioning ourselves for the eventual conduct of the longer term effort. And perhaps that's where a field study belongs, when we have more resources or when we are undertaking a longer term research program.

But nevertheless, priorities do play an important role in adjusting the agenda for, or setting the agenda for research; and, quite frankly, the Indoor Air Research Program is planned, and has been planned differently than the rest of our research program. Most of our research is very policy relevant. And I think one of the good pieces of news that we can bring to you this particular hearing is that under the championing of our Administrator, Mr. Thomas, he has a policy group in the office of air; and working collaboratively with that organization, I think that we will be able to design a research program that is very policy relevant, and it will be in accord with agency mission or agency policies.

I believe also that in these past 2 or 3 years that I have seen considerably more success having taken place with regard to the Interagency Committee on Indoor Air Quality. I believe there is certainly evidence that more of our scientists are talking to scientists of other agencies, and so consequently I look upon that as being a very, very favorable outcome. And there is certainly a lot more joint funding of projects between us and other agencies.

Well, we have operated from the perspective of a fishbowl from the outset, letting anybody come in and review our program. And quite frankly, sometimes that leads to some conflicts. And there have been a lot of conflicts that you have probably read about and that you were alluding to. We are uncomfortable about those sorts of things; but, nevertheless, what is most important to us is having a strong scientific basis, a strong scientific program.

A recent Science Advisory Board panel just met, or recently a science advisory board panel met in North Carolina to review the program, and I think Dr. Jan Stolwijk will be able to talk to you in a little more detail about that. But his initial observations to me were that our science was of high quality, and that's important to us in the office of research.

Well, that concludes my brief summary, as per your request, Mr. Chairman, and I'm either available for questions or perhaps to invite Mr. Myers to talk about the radon program.

[The prepared statement of Mr. Ehreth follows:]

PREPARED STATEMENT OF DONALD J. EHRETH, ACTING ASSISTANT ADMINISTRATOR FOR RESEARCH AND DEVELOPMENT, U.S. ENVIRONMENTAL PROTECTION AGENCY

Good afternoon, Mr. Chairman and Members of the Subcommittee. My name is Donald J. Ehreth, and I am the Acting Assistant Administrator for Research and Development of the U.S. Environmental Protection Agency (EPA). I am pleased to be here to present a statement about the status of our indoor air quality research in the Agency.

On the average, Americans spend the majority of time during the day indoors. Indeed, if the time spent inside automobiles, buses, and trains is excluded, Americans probably spend very little of their time, on the average, outdoors. In addition, existing field data on the concentrations found indoors indicate that indoor levels for many pollutants of concern to EPA are generally higher than outdoor levels. For the most part, however, the nature of the indoor air quality issue has not been adequately characterized. Our research seeks to better define and understand the nature of the indoor air quality levels and their possible effects.

We appreciate this opportunity to describe our indoor air quality research program since on September 4, 1986, a peer review of our research program was conducted by a special panel of experts under the sponsorship of the Agency's Science Advisory Board (SAB). We have paused in the implementation of our current research program in order to assess what we know and don't know about the public health impacts of indoor air pollution and consulted this expert peer panel convened by the SAB to provide us with views about the approach and the general course of our planned research activities.

This peer review is one in a series designed to provide the Agency's Deputy Administrator with an outside review of EPA research while it is in programs. Panel reports provide independent expoert views about research programs.

The presentation to the SAB panel consisted of three elements: (1) a description of ongoing indoor air research projects; (2) a limited exploratory field study; and (3) a reassessment of the state of scientific knowledge concerning indoor air pollution.

Following are some of the more interesting ongoing research projects in our indoor air research program. They are grouped in three major areas of research:

(1) Methods development and studies to characterize indoor air pollution exposures:

Improvement in sensitivity and specificity of NO_2 monitors to permit more reliable operation in field studies.

Evaluation of methods to monitor organic chemical compounds including formaldehyde and polynuclear aromatic hydrocarbons.

Evaluation and improvement of survey questionnaires for field studies.

(2) Methods development and studies to determine health effects of indoor air pollution:

Evaluation of continine as a biological marker for exposure to tobacco smoke in laboratory trials and field studies.

Evaluation of lung function tests to obtain information about NO_2 exposure while cooking with a gas range.

Investigation of neurobehavioral changes due to indoor exposure to volatile organic compounds.

(3) Methods development and studies to characterize and quantify the sources of indoor air pollution and to mitigate those sources:

Design and standardization of chamber test procedures for emission testing of building materials and consumer products.

Emission testing of unvented kerosene space heaters as an example of combustion appliances to develop optimum test procedures and to determine if emissions are carcinogenic or mutagenic.

Development of an emission data base for building materials and consumer products.

Demonstration of radon mitigation by venting in existing homes in the Reading Prong.

The ongoing EPA research program as well as the reassessment of the state of scientific knowledge and the development of a reviewed detailed plan for research in FY 1987 is entirely consistent with the research strategy that was prepared for Congress in January 1985 in coordination with the Interagency Committee on Indoor Air Quality (CIAQ). The status of implementing this strategy has been described in detail in response to an inquiry about CIAQ research activities from Congressman Boland.

Implementation of the CIAQ research strategy has improved our awareness about indoor air quality. In collaboration with CIAQ member agencies, we have: improved field survey and health effects questionnaires, established procedures to test building materials and combustion appliances in chambers to determine if they are important sources of indoor air pollution, and established a data base for pollutant emissions. We have contributed to ongoing health effects studies concerning indoor air quality to better evaluate the risks associated with exposure of these pollutants indoors. These indoor air research efforts have been augumented with other supplemental research projects and base program activities to balance our research program to achieve the goals of the CIAQ strategy.

As part of our CIAQ commitment, we took the lead on exposure monitoring, instrument development and evaluation, and field study design in response to peer review recommendations, interest among CIAQ members and Congressional direction. We conducted pilot field studies to test monitoring instruments and survey questionnaires. We designed large-scale field surveys for combustion products and organic compounds to provide a national estimate of exposures to indoor air pollutants. However, reviews, of these proposals prompted concerns about the utility and need for such a survey. These concerns have prompted our action to reassess the direction of the research program to ensure that it will be tailored to meet Agency needs and be responsive to Congressional direction.

Development of our future research plans will be coordinated with our Office of Air and Radiation (OAR). OAR has recently initiated a policy coordination and development process in EPA and will commit base program resources to address indoor air quality issues for the first time in FY 1987. OAR will be the lead office for developing and coordinating program strategies for indoor air.

OAR will act as the client program office for ORD's indoor air research program. They are now developing the analytic basis for identifying and prioritizing program responses to indoor air. This analysis will be based in large measure on the results of past and ongoing research which we are making available to them. We are developing working arrangements with OAR to insure that program and research strategies are jointly planned and coordinated through the research committee process.

This concludes my prepared statement, Mr. Chairman. I would be happy to respond to any questions.

Mr. **Scheuer**. Very good. Thank you very much, Mr. Ehreth. Now we will hear from Sheldon Meyers, Deputy Director of the Office of Radiation Programs. He'll chat with us informally for 5 minutes.

Mr. **Meyers**. Mr. Chairman, members of the subcommittee: I, too, will summarize these remarks, and I will only be talking about radon, rather than indoor air in general.

In the recent report that the General Accounting Office put out, they surveyed a number of States to determine their views on appropriate Federal radon activities. I'm pleased to say that the GAO survey identified program elements that were very, very similar to the program we had already put underway.

While EPA has been very active in addressing the radon problem, it has certainly not been the only agency involved. The Department of Energy is conducting research as part of its radiation and energy research activities. Also, the U.S. Geological Survey, the Department of Housing and Urban Development, and the Center for Disease Controls also have roles to play.

These and other Federal agencies have been participating in two interagency committees to review research needs, to identify key policy questions and to coordinate the objectives of our various programs. Through these committees and other informal relationships, EPA staff is in constant contact with other Federal agencies to coordinate our respective efforts. This is important because we have heard that they were disparate programs, and these are two mechanisms that we have been using to make certain that the efforts are coordinated amongst Federal agencies.

Now we have divided our radon action program into four elements, and these are: Problem assessment, in which we establish standardized measurement methods and assess the extent of the radon problem; mitigation and prevention, where we conduct extensive research and gain experience with methods of reducing radon levels in existing and new homes; capability development, a very important part, which supports State programs to develop a capability to measure and diagnose radon problems; and lastly, public information, which is a very critical part of the program where we disseminate useful information to the public at large and to State and local organizations.

Within each of these areas the agency is conducting an array of projects involving both research and operational assistance to States. Although our initial initiatives have been targeted at the Reading Prong area within Pennsylvania, New York and New Jersey, the radon action program is also laying the groundwork for identifying and dealing with similar problems elsewhere in the country.

Let me summarize by saying that we have done a lot in the past 18 months to respond to this potential health threat, major elements of the program are now in place, and much of the success of our effort rests on the development of a strong Federal-State partnership. When the program is fully implemented at all levels, Federal, State and local, we will be closer to our goal of significantly reducing the health risks from radon.

Thank you. I, too, will be available for answering questions.

Mr. SCHEUER. Thank you very much.

[The prepared statement of Mr. Meyers follows:]

PREPARED STATEMENT OF SHELDON MEYERS, DIRECTOR, OFFICE OF RADIATION PROGRAMS, U.S. ENVIRONMENTAL PROTECTION AGENCY

Mr. Chairman and Members of Subcommittee: good afternoon. My name is Sheldon Meyers. I am the Director of EPA's Office of Radiation Programs. I am pleased to be able to discuss with you this afternoon some of EPA's radon activities.

Although there has been growing awareness among scientists over the past few years that elevated levels of naturally occurring radon in homes might cause significant health risks, it was just recently that extremely high radon levels were found to be widespread. Not until 1985, after the discoveries of extremely high levels in the "Reading Prong" area of Pennsylvania and the thousands of homes involved in that area alone, did the severity of the indoor radon problem begin to be fully appreciated by scientists, government officials, and the public.

As EPA began to respond to these discoveries, we quickly realized that, while we knew a great deal about the problem, the actions to reduce risks needed to occur at the State and local level. The public needed information about specific situations, information that could not be fully developed or discussed with them by Federal officials far away from the region of concern. With this in mind, we spoke to many scientists and State and local officials to determine what the major scientific and public information needs were, and how this information could best be provided to the public. We found the following needs on just about everyone's high priority list:

1. The need to assess the size of the problem, determine what parts of the country are affected, and how to predict where radon will be a problem.
2. The need to standardize measurement methods.
3. The need to evaluate the competence of firms and laboratories that offer measurement services.
4. The need to develop cost-effective ways to reduce radon levels in existing and new homes.
5. The need to develop technical expertise within State and local governments, as well as in the private sector.
6. The need to develop and publish guidelines explaining the health risks associated with various levels of radon, and offering recommendations on what actions should be considered by homeowners.
7. The need to develop effective communication tools to present health concerns and recommended solutions to the public.

In response to these and other needs, EPA developed the Radon Action Program. This non-regulatory program includes both research and operational components. Our overall approach has been to work in partnership with the States to develop a full range of tools that citizens will need to reduce the health risks of radon.

In their recent report on indoor radon, the GAO surveyed a number of States to determine their views on appropriate Federal radon activities. The GAO survey identified program needs similar to the seven we identified at the outset of the Radon Action Program. Most of the States supported the current Federal approach of conducting research and providing technical assistance, and specifically mentioned some activities they want the Federal Government to be involved in. These activities include radon mitigation research, establishing national measurement standards and remedial action guidelines, and instituting a program to evaluate radon detectors and measurement films. Consequently, we believe the GAO report confirms that we have tailored our program to meet the most important needs of the States and the private sector.

While EPA has been very active in addressing this problem, it has certainly not been the only agency involved. The Department of Energy is conducting research projects as a part of its radiation and energy research activities. Also, the U.S. Geological Survey, the Department of Housing and Urban Development, and the Centers for Disease Control all play important roles in dealing with radon. These and other Federal agencies have been participating in two interagency committees to review research needs, to determine key policy questions, and to coordinate the objectives of our various programs. Through these committees and other informal relationships, EPA's staff is in constant contact with the other Federal agencies to coordinate our respective efforts.

We feel that EPA's Radon Action Program has made substantial progress toward meeting most of these needs. Programs are already in place to conduct mitigation research, and standardized measurement protocols have been issued for a number of radon/radon decay product measurement devices. Also, the Radon Measurement Proficiency program is in place and has issued two periodic reports to the States listing those firms that have demonstrated competence in measuring radon or radon decay products.

Another important part of our Program is the development of information for the general public that explains the complex technical and health concerns associated with indoor radon. We recently issued two brochures, "A Citizen's Guide To Radon: What It Is and What to Do About It," and "Radon Reduction Methods: A Homeowner's Guide." These two publications have been provided to the States for distribution to concerned citizens. These brochures have been well received by the States and the public. Many States are now preparing to print additional copies.

The needs we believe the Radon Action Program should address can be divided into four elements:

Establishing standardized measurement methods and assessing the extent of the radon problem (problem assessment);

Conducting extensive research and gaining experience with methods of reducing radon levels in existing and new homes (mitigation and prevention);

Supporting State programs to develop a capability to measure and diagnose radon problems (capability development); and

Disseminating useful information to the public (public information).

Within each of these areas, the Agency is conducting an array of projects involving both research and operational assistance to States. Although our initial activities have been targeted at the Reading Prong area within Pennsylvania, New York, and New Jersey, the Radon Action Program is also laying the groundwork for identifying and dealing with similar problems elsewhere in the country. To give the Subcommittee an overview of our program, I would like to summarize our recent accomplishments and future plans in each of the four areas.

PROBLEM ASSESSMENT

National and State Surveys.—Our program will include a national survey and assistance to State survey efforts to obtain statistics of radon levels in homes, to identify areas where elevated radon levels are likely to occur, and to identify factors that may influence radon levels. Some 15 States already have surveys underway or are planning them. The design for our national assessment is currently being reviewed by EPA's Science Advisory Board. We will also evaluate data that are available from ongoing private sector activities to facilitate our efforts.

Measurement Methods and Procedures.—To insure that radon measurements from various sources are accurate and comparable, we published seven interim protocols, or methods, for measuring indoor radon on February 25, 1986. Guidance on the use of these methods for various purposes is now being prepared and should be released within a few weeks. We anticipate developing additional standardized measurement methods as needed. For example, we are looking into developing standardized procedures to estimate exposures for the various epidemiological studies that are being initiated.

Quality Assurance.—As mentioned, the Agency has established a national Radon Measurement Proficiency (RMP) program to evaluate the capability of private firms and other organizations involved in measuring indoor radon levels. Participation in the program is voluntary, but States may use the proficiency evaluations as a basis for certification or other action aimed at assuring competency. Interest in the program is growing rapidly; while only 35 firms participated in the first round, 90 firms participated in the second round. More than 200 requests for applications to the third round have already been received. EPA will periodically issue lists of the organizations that have demonstrated adequate proficiency in measuring radon. The most recent report from our RMP program was released on September 2, 1986. (A copy of this report is attached).

Land Evaluation.—EPA is working with the U.S. Geological Survey to determine potential uses of geological and other relevant data to help identify areas where elevated radon levels could be expected are being explored jointly with the U.S. Geological Survey. Alternative soil gas measurement techniques are also being evaluated.

MITIGATION AND PREVENTION

Radon Mitigation Research Program.—The Agency is developing and demonstrating low-cost methods for reducing radon concentrations inside homes. The initial 18-home demonstration program in Pennsylvania has been completed. A manual based on its findings has been published for use by contractors and competent homeowners. Mitigation techniques are being tested in additional homes in Pennsylvania and New Jersey, with another project now getting underway in New York.

House Evaluation Program.—We are also supporting a radon diagnosis and mitigation project involving a number of homes with elevated radon levels in the Reading Prong area of Pennsylvania. State employees, supported by EPA staff and contractors, visit selected homes, determine where and how radon is entering the homes, and give the homeowners information on alternative ways of reducing the radon levels. The homeowners themselves are then responsible for deciding what action to take and for financing the mitigation effort with assistance from the State as available. This program will help generate data at a moderate cost to EPA on the effectiveness of various control measures, and will also help build expertise within State and local governments on how to diagnose problems in homes and advise homeowners of possible solutions. By the end of this fiscal year, 80 homes will have been evaluated under this program, with another 80 evaluations scheduled for FY 1987.

New Home Design and Construction.—During the next fiscal year, the Agency plans to identify and evaluate new home design and construction techniques that would avoid or minimize radon problems. Knowledge from this effort could be incorporated into model building codes for State and local use.

CAPABILITY DEVELOPMENT

Mitigation Training.—We have developed a course to train Federal and State government employees to measure and diagnose radon problems and give homeowners information on mitigation alternatives; we are also making the course available to audiences of private contractors as arranged for by interested States. By the end of September, 20 training sessions will have been conducted. These courses have been concentrated primarily in the Reading Prong area, but plans are underway to offer training in other parts of the country as well.

Measurement Training.—The Agency routinely helps States and private firms by providing technical advice on radon measurement methods and by offering calibration exposures so that firms may evaluate their own capabilities. EPA also provides assistance to States that wish to establish their own measurement programs.

Private Sector Outreach.—The radon program is not just a Federal effort, but a combined Federal, State, local, and private sector effort. Numerous associations, industry groups, and other private sector organizations have expressed interest in how they can use their resources and activities to help deal with this public health problem. We will continue to work with a wide range of these groups to help them prepare their members to assess and mitigate radon problems. National and regional groups of health organizations, home builders, realtors, and mortgage lenders are among those involved.

PUBLIC INFORMATION

Guidance.—The centerpiece of our public information efforts to date has been the release of the two pamphlets I mentioned earlier which describe what radon is, how it threatens health, how it can be measured, and what measures can be taken to reduce levels in homes. The Agency has also recently published a technical manual on radon mitigation techniques that provides additional detail.

To summarize, we have done a lot in the past 18 months to respond to this potential health threat. Major elements of our program are now in place, and much of the success of our efforts rests on the development of a strong Federal-State partnership. When our program is fully implemented at all levels, Federal, State and local, we will be closer to our goal of significantly reducing the health risks of radon.

Thank you.

Mr. **Scheuer**. Thank you very much.

Now we will hear from Dr. Charles DeLisi.

Dr. DeLisi. Mr. Chairman, members of the subcommittee: it is a pleasure to be here and to describe the Department of Energy's program in radon research and indoor air quality.

The Department of Energy has had a program in radon research for well over a decade, and it stems largely from our, or from the Department's responsibilities for understanding the effects of low levels of radiation. More recently the Department's interest has stemmed from energy conservation methods and trying to understand the effects and possible buildup of low levels of radiation and other indoor pollutants.

There are basically three organizations within the Department responsibile for its program. One is the Bonneville Power Administration, and they have been studying the effects of weatherization in the Northwest on buildup of contaminants indoors. There is the Office of Conservation and Renewable Energy, which Mr. Millhone, at my right, is representing; and the Office of Energy Research of which I am the Associate Director for the Office of Health and Environmental Research.

There are two broad components to the Department's program. One is directed at doing the research which would lead to the development of the best and most cost-effective mitigation measures. The other is directed at defining more precisely the uncertainties that we're now faced with with respect to low levels of radon.

We are also very much aware of some of the coordination problems. We cochair the Committee on Indoor Air Quality with the EPA, as was just described. In addition, there is an important committee in the Office of Science and Technology Policy which is responsible for coordinating radiation research. There is a radon panel which EPA and DOE are both part of. And we have also developed a formal agreement with the Committee for European Communities to assure that there is no overlap in synergism when that becomes possible.

That concludes my statement. I will be glad to answer any questions.

[The prepared statement of Dr. DeLisi follows:]

Prepared Statement of Charles DeLisi, Associate Director for Health and Environmental Research, Office of Energy Research, U.S. Department of Energy

Mr. Chairman and Members of the Subcommittee, I appreciate the opportunity to appear before you today to provide information on the Department of Energy's research related to residential radon contamination and indoor air quality.

INDOOR AIR QUALITY RESEARCH PROGRAM

In addition to its historic mission and expertise in the area of radiation effects research, the Department of Energy (DOE) conducts a variety of activities related to indoor air quality (IAQ) in support of DOE policy to encourage the use of advanced energy conservation measures while maintaining a safe and healthful indoor environment.

Five DOE offices support research or conduct other activities in the area of indoor air quality: Energy Research; Conservation and Renewable Energy; Environment, Safety, and Health; Nuclear Energy; and the Bonneville Power Administration.

OFFICE OF ENERGY RESEARCH

The Department of Energy and its predecessor agencies have sponsored much of the fundamental research in the U.S. aimed at defining and reducing radiation exposures and the consequent risks in both occupational and environmental settings. The Biological and Environmental Research Program within the Department of Energy, initiated radon research to address concerns about occupational exposures in uranium mines and mills and other industrial activities. Since 1975, as more information became available on the magnitude of environmental radon exposures, emphasis has increasingly been placed on indoor radon studies.

The Office of Energy Research's (ER) radon research program has, among its accomplishmets, contributed to: improved radon measurement techniques, e.g., the charcoal canister technique; our understanding of lung deposition as a function of particle size; and the complex relationship between radon and smoking.

In response to increasing concerns about indoor radon exposures, the Office of Health and Environmental Research (OHER), within the Office of Energy Research, has developed a draft multidisciplinary radon research plan. The starting point for developing OHER research priorities has been consideration of the degree of risk associated with existing exposures and the availability of options for reducing this risk. In addition, DOE has a special interest in the possible future impact of continued and increased energy conservation on radon exposures and in the reduction of future radon exposures by appropriate changes in new building technology.

Research cooperatively funded by ER and CE has shown that the dominant source of indoor radon into most structures is radon in the surrounding soil. Moreover, the flow of the soil gas across the building envelope has been shown to be driven primarily by pressure differentials produced by thermal gradients, wind effects, and building ventilation. An important goal of the proposed research program is the development of a detailed model of both radon availability in the soil and radon transport into structures. Such a model could then be applied to the evaluation of potential mitigation techniques that focus on the reduction of the radon entry rate into structures, and would provide a basis for estimating the probability that high indoor radon levels would be found in particular areas. A better understanding of the complex relationship between pressure differentials and ventilation rates is also necessary to determine the impact of energy conservation on the radon risk.

A second research goal is to identify the respiratory cells at risk from radon exposure and to improve our capability to quantify the dose to these cells. New and improved radon measurement methodologies are currently being developed and evaluated, and this effort along with appropriate quality assurance programs will be continued. Environmental influence on radon decay product properties, particularly concentrations, charge, and particle size, also needs to be better understood. Models of these phenomena can be applied to the evaluation of air-cleaning techniques. Finally, a better understanding of particle deposition patterns in the lung and biological removal mechanisms in the respiratory tract is needed to accurately and quantitatively estimate dose. This capability is especially important in the interpretation of data from controlled animal experiments to human health effects data.

A third goal of ER's radon research program is to conduct appropriate epidemiological studies to quantify the relationship between lung cancer incidence and environmental radon exposure. A major study of this type has recently been initiated in eastern Pennsylvania. Such studies are unlikely to define dose-response relationships over the entire range of human exposures, but may provide reasonable estimates of risk at high exposure levels and suggest possible relationships between radon exposure and smoking in the induction of lung cancer.

The fourth and last goal, probably the most important one in the long run, is the development of a fundamental understanding of the role of radon in the induction of lung cancer. A detailed model of the development of lung carcinogenesis would be extremely valuable in providing a firm basis for inferring the dose-response curve and determining the interaction between radon and smoking in lung cancer industion. Due to the immediate importance of this goal, it deserves the full attention of the scientific community.

In response to OHER's plan to expand its radon research program, over 30 new research proposals have been received and are currently under review, and a comprehensive radon research program will be in place in early FY 1987. Decisions on individual projects will take into account relevant work being sponsored by other DOE offices, EPA and other Federal and State agencies, and by the Radiation Protection Program of the Commission of the European Communities. Thus, the overall program will be a multi-agency effort, with close collaboration and continuing communication required to ensure timely information and technology transfer.

OFFICE OF CONSERVATION AND RENEWABLE ENERGY

Research conducted by the Office of Conservation and Renewable Energy (CE) is devoted primarily to determining the relationship between indoor air quality and energy conservation. To develop this information, CE performs indoor air quality research to (1) characterize indoor pollutant sources including radon; (2) develop, test, and validate measurement techniques and models to quantify indoor pollutant levels and ventilation rates; and (3) develop, test, and evaluate energy efficient IAQ control techniques.

Prior CE research has been devoted to understanding radon building entry mechanisms; air-to-air heat exchangers; and the relationships among indoor air quality, infiltration, and ventilation in buildings.

As mentioned earlier, CE research cooperatively funded with ER has demonstrated that the primary source of indoor radon is from surrounding solid, and that the predominant building entry mechanism is from pressure-driven flow. Current CE research, cofunded in part with the Bonneville Power Administration and the Environmental Protection Agency, is devoted to quantifing the factors that influence radon entry and developing energy efficient mitigation techniques to reduce indoor radon levels. The goal of this research is to provide the information necessary to develop a capability to estimate indoor radon concentrations in both existing and planned residences, and providing the basis upon which energy efficient design and retrofit techniques can be developed for the construction of low radon homes.

The effectiveness, energy efficiency, and cost effectiveness of air-to-air heat exchangers has also been an area of CE research. Similar studies are currently being performed on air-to-water heat pumps. Preliminary research results indicate that these devices may be a more energy efficient means of providing residential ventilation to reduce indoor pollutant levels, including radon.

Finally, CE research in large buildings has demonstrated that these buildings which experience periods of under- or over-ventilation will compromise quality or energy conservation. In FY 1986, research was initiated which simultaneously measured indoor pollutant levels, including biologically active aerosols, and ventilation rates. Two additional buildings will be measured in FY 1987.

OFFICE OF ENVIRONMENT, SAFETY, AND HEALTH

Within the Office of Environment, Safety, and Health (EH), there are three activities related to indoor air quality. The first, the Aerial Measuring System, is used to prepare background radiometric maps of nuclear facilities in the U.S. These serve as the baseline for rapidly assessing the extent and severity of any radiological accident. Another activity within EH includes the synthesis of currently available information on specific indoor air quality topics into information handbooks for use by State and local public health officials, policy analysts, builders, architects, engineers, homeowners, and consumers. Handbooks on combustion sources and radon have been completed; a handbook addressing the impact of building system characteristics on indoor air quality will be completed in FY 1987. Finally, EH is cooperatively funding with CE and the Consumer Product Safety Commission (CPSC) a combustion sources exposure model to determine the distribution of indoor concentrations of combustion pollutants in single-family dwellings.

OFFICE OF NUCLEAR ENERGY

The Office of Nuclear Energy (NE) is responsible for DOE's Remedial Action Program. The DOE Remedial Action Program has been organized into four separate subprograms. The Grand Junction Remedial Action Program is responsible for correction of elevated indoor radon levels associated with the disposition of uranium mill tailings in the Grand Junction, Colorado area. Cleanup of an estimated 600 structures is scheduled for completion in 1987. The Uranium Mill Tailings Remedial Action Program is responsible for stabilizing 24 designated uranium mill tailings sites in 9 western states and one at Canonsburg, Pennsylvania, as well as clean up of an estimated 4800 contaminated properties in the vicinity of processing sites. The Nuclear Regulatory Commission (NRC) is responsible for approval of remedial action plans and licensing sites which meet criteria established by the ERA. The Formerly Utilized Manhattan Engineering District/Atomic Energy Commission Sites Remedial Action Project is responsible for the identification and disposition of facilities that were formerly used in government nuclear programs and then released for unrestricted use, but which have radioactive contamination above current limits. Work has been completed on 6 of the 29 currently designated sites. The Surplus Facilities Management Program is responsible for disposition of radioactive government-owned facilities that are no longer needed for government programs. Thirty-four civilian projects at 15 sites are currently in inventory to be decontaminated and decommissioned.

BONNEVILLE POWER ADMINISTRATION

Bonneville Power Administration (BPA) research devoted to indoor air quality focuses upon evaluation of: the impact of energy conservation measures on indoor air quality in new and retrofitted homes; energy efficient mitigation measures, including carbon dioxide based ventilation control systems; and indoor pollutant measurement techniques. Radon and formaldehyde concentrations are currently being measured in new energy efficient homes built to meet the Model Conservation Standards proposed by the Northwest Power Planning Council. Also, energy efficiency improvements and formaldehyde concentrations are being evaluated in manufactured homes.

BPA is also involved in the dissemination of indoor air quality literature through its residential weatherization program. Supported by BPA field studies, the Weatherization Environmental Impact Statement, completed in the fall of 1984, found pollutant levels in Northwest homes to be generally low, and concluded that when consumers are well-informed about indoor pollutants and their associated risks, they can best determine if indoor air pollution is a problem in their own homes. BPA distributes consumer information and offers free radon monitoring to all participants through utilities and state agencies implementing the program. If radon levels are found in excess BPA's action level of 5 picocuries per liter, BPA shares the cost of installing air-to-air heat exchangers provided that participants adopt house tightening measures.

RESEARCH COORDINATION

DOE also coordinates its research on indoor air quality with the other Federal agencies conducting research in this area. DOE cochairs the interagency Committee on Indoor Air Quality (CIAQ) as well as the CIAQ Radon Workgroup and several other workgroups. The department is also represented on the Committee for Interagency Radiation Research and Policy Coordination (CIRRPC). DOE provides information and expertise to technical and professional societies such as the American Society of Heating, Refrigerating, and Air-Conditioning Engineers (ASHRAE), Air Pollution Control Association (APCA), and ASTM. In addition, DOE participates in international cooperative research planning on radon with the Commission of European Communities (CEC) and supports and participates in the IAQ activities of the International Energy Agency (IEA).

The complexity of the indoor air quality issue, with its potential public health and energy implications, requires a concerted, integrated, and high quality scientific research effort. This effort is necessary to reduce the large scientific uncertainties that now exist in these areas. To meet these needs in a timely and balanced manner, the DOE researh program as provided for in the President's FY 1987 budget request calls for the continuation of DOE's ongoing indoor air quality research and an expansion of its radon research effort to specifically target those areas of greatest uncertainty and potential public health impact.

That concludes my prepared statement. I would be happy to answer any questions you have at this time.

Mr. SCHEUER. I am first going to ask Mr. Ehreth and then I am going to ask Mr. DeLisi, speaking for your two respective agencies.

Oh, let me just cut short for a moment and say we've been joined by two members. First of all, we've been joined by Jan Meyers—Congresswoman Jan Meyers of Kansas—a very hardworking and dedicated member of this subcommittee who I don't think has ever missed a single meeting of this subcommittee, if my memory serves. She is a very terrific lady.

And we've been joined also by Gus Yatron of Pennsylvania, who is a very dedicated and hardworking Congressman on the Foreign Affairs Committee, and who has a particular concern for the problem of radon became he comes from a part of Pennsylvania that has a great deal of it, and he is providing a great deal of leadership on the matter in his home State.

Let me ask, Mr. Ehreth, first, what have you learned so far from your research that would be of interest to us, and of interest to the public? What have you learned on the bottom line?

Mr. EHRETH. Most of our research to date has been aimed at developing the instrumentation, laying the fundamental framework for a longer term program in the out-years. What was missing from—at the point when we embarked upon indoor air research was the instrumentation that was necessary to be able to measure and monitor some of the organic, some of the particulate matter and some of the combustion by-products. We have been able to develop those kinds of instruments.

In the area of health——

Mr. SCHEUER. Have you done any applied research at all that would give the public and the Congress some clues as to the dimensions of the problem?

Mr. EHRETH. Yes.

Mr. SCHEUER. What the problem is and what we ought to do about it?

Mr. EHRETH. We have subsequently learned, or also learned during this period of time that volatile organic compounds are found in the indoor environments at concentrations 8, 10 and more times higher than are found out of doors. We have found, as I indicated earlier, that cigarette smoking or environmental tobacco smoke that is talked about, termed or called in our agency, is probably the most potent mutagen found in indoor environments. We also know that there is another by——

Mr. SCHEUER. Is it more so than radon? More carcinogenic than radon?

Mr. EHRETH. No. I would say radon—my understanding is radon is still the leading chemical in the way of lung cancers and things of that nature. But everyone also seems to agree that when exposed to both radon and cigarette smoke there is some synergistic effect there that enhances, or shall we say adds to the risks associated with lung cancer.

Mr. SCHEUER. Yes.

Mr. EHRETH. The other thing that I think our research program has that we have transferred already to the public in Pennsylvania and in New Jersey, and in other places we're working, is in the area of radon mitigation technology.

Excuse me, sir. You were going to ask me something?

Mr. SCHEUER. No.

Mr. EHRETH. Oh, I'm sorry.

Mr. SCHEUER. I might have suggested to Gus Yatron that he better tell his people at home there to stop smoking; that they have a lot of radon which they can't do much about fast. The one thing they can do is stop this symbiotic effect by stopping their smoking.

Mr. EHRETH. We often give that as one of the first pieces of advice, as a matter of fact, to stop smoking also if you have already detected radon.

But more importantly, I think we have been able to give people practical advice and guidance in how to mitigate. First of all, how to diagnose if they have a radon problem. And I think that's come about as a—it's a good model, shall we say, for how the Office of Air and Office of Research and other agencies can work together on fostering solutions to these national problems. And I think that radon is an excellent model which has demonstrated that ability.

And in this case we have developed those sorts of mitigation techniques, and we have manuals. I have some information here I'd like to just submit for the record, and I'm sure Mr. Meyers has a manual on the radon outputs, also, products that we've recently had printed up—material. We would like to submit that for your information, for the committee's reviews.

But I think, in short, those are the types of things that we've been able to do. We also found out in our source characterization studies that there are very few things found indoors that don't outgas some organic compound or, in case of combustion systems, produce some sort of byproduct of combustion; and all these lead to some sort of sensation on some subsets of the population.

What has been very difficult to do up to this point in time is begin to associate cause and effect, or source and receptor. This is the effect, now what's the source, specific source related to that effect? And that is where I think the research program will be heading more toward in the future, and some of the advice that we've received from the SAB panel seems to indicate that if we deal more on a balanced program, balancing our efforts in source characterization, in some field studies, limited field studies, in health effects, and mitigation, perhaps we would have a lower—better balanced approach. The next time we sit down with you we'll be able to answer that question more specifically by saying here is what we know and here is what you can do about it. Because quite frankly that's what we're aiming to do.

We're trying to provide answers to the homeowner, to the building engineer, saying—who has the following questions: Do I have a problem? And if I have a problem, what do I do about it? That is the driving force behind our research program.

Mr. SCHEUER. Well, I know that's behind the research program. What I did ask you is what have you found out to date. We would like to know if the research has produced any guidance for the American people.

But my time has expired, and I want to share the time equally with my colleagues. I do want to recognize we've been joined by our colleague Paul Henry of Michigan, and our colleague Bob Smith of Vermont, and at this point I'd like to recognize for a statement our distinguished colleague Gus Yatron of Pennsylvania, who is a distinguished guest here. He is not a member of this committee, but he is a distinguished member of the Foreign Affairs Committee and always welcome here.

Gus.

Mr. YATRON. Thank you very much, Mr. Chairman. I want to say that I am pleased to be a part of this hearing today, and I appreciate Chairman Scheuer asking me to participate in it.

I represent part of the Reading Prong and the town of Boyertown, PA is in my congressional district. Boyertown is where the problem of indoor radon was first discovered. That was in December 1984. And I, along with many other people in our area, have been grappling with this issue ever since.

I have a longer statement, and in the interest of time I would like to make a request that it be added to the hearing record.

This afternoon I would like to make just two points. First, I think we need to make homes that are contaminated with extremely high levels of radon our highest priority. In these cases the cost of cleanup can be higher and there is a more imminent danger to the health of the residents in our area and around the country. I would like to see some direct assistance to the homeowners in this category.

I would also like to make a comment about the debate between radon and indoor air in general. There are those who think it is shortsighted to address radon by itself; that the problem is really one part of the general indoor air quality problem. Well, I tend to agree, but I believe that radon is the indoor air pollutant that poses the greatest threat to health.

Moreover, since it is not a manmade pollutant, it has special qualities in terms of how it—how we should address it in law, policy, and in governmental programs. I certainly would not oppose its incorporation into a general indoor air program, but I want to see this problem solved as soon as possible. I hope this policy debate will not slow our efforts. The important point is to give the Federal Government the legislative authority it needs to give this problem the priority it warrants.

Finally, I want to commend my distinguished colleagues, Chairman Scheuer and the ranking minority member, Claudine Schneider, as well as all the other members of the committee for their work in this area. They have been fighting for greater attention to indoor air quality long before we knew indoor radon could be a hazard. I also commend them for today's hearing and for the one that they held last October. This Science Subcommittee has been very responsive and willing to help address a very complicated issue, and I want to commend my colleagues and all the members of the subcommittee for their efforts.

Thank you, Mr. Chairman.

[The prepared statement of Mr. Yatron follows:]

PREPARED STATEMENT OF HON. GUS YATRON

I am pleased to be part of this hearing today and I appreciate Chairman Scheuer asking me to participate in it. I represent part of the Reading Prong and the city of Boyertown, Pennsylvania is in my Congressional District. The problem of indoor radon was discovered in Boyertown in December, 1984 and I have been grappling with the issue ever since.

Initially, my efforts focused on helping one family find a way of making their home livable again. At first, I thought there would be some program at the federal level that could help pay for the cost of a mitigation device to reduce the radon level. Shortly after the discovery, Pennsylvania's Department of Environmental Resources started a testing program that soon uncovered thousands of other homes along the Reading Prong which had very high levels of radon.

The road from that point to this one has not been an easy one for many of my constituents. While we have made some progress in the nearly two years since the discovery, a General Accounting Office study published in July stated that federal efforts to addresss this very serious, national problem were uncoordinated and inconsistent.

I introduced a bill on July 17th incorporating many of GAO's recommendations and currently the measure has 51 cosponsors. Many of the provisions of the bill have been incorporated into the Superfund reauthorization. Passage of Superfund may bring federal efforts into better focus.

Fiscally, this is a very difficult time for the federal government to be taking on another and possibly very costly responsibility. Our resources are limited and I think this is all the more reason why our efforts to address the problem h[illegible] to be very precise and well-organized.

I know there has been some disagreement about the action level EPA set for radon. Some think .02 wl may be too low a standard to set and that the number of homes at this level could well be around 8 million. I hope the health effects study DOE is conducting will be able to clarify this for us. In the meantime, I think our priorities must be the following:

EPA should help states identify potential hot spots and the agency should provide as much technical assistance as possible to states on testing and mitigation.

EPA should be given necessary funds to develop low cost, effective mitigation devices so that when homeowners find out they have high levels of radon in their homes, they have some proven way of reducing it.

I would like to see us in some way provide assistance to homeowners whose levels are at astronomically high levels. In these cases, the cost of cleanup can be higher and there is a more imminent danger to the health of the residents. There may be 100,000 homes in the nation at 1 working level and we should try to make these safe as soon as possible.

Finally, I would like to make one comment about the debate between radon and indoor air in general. There are those who think it is shortsighted to address radon by itself, that the problem is really one part of the general indoor air quality problem. Well, I tend to agree but I believe that radon is the indoor air pollutant that poses the greatest threat to human health and that since it is not a man-made pollutant it has special qualities in terms of how we should address it in law, policy, and in governmental programs. I certainly would not oppose its incorporation into a general indoor air program but I want to see this problem solved as soon as possible. I hope this policy debate does not slow our efforts. The important point is to pass the Superfund amendment and give the federal government the legislative authority it needs to give this problem the priority it warrants.

Lastly, I want to commend my distinguished colleagues, Chairman Scheuer and Representative Schneider for their work in this area. They have been fighting for greater attention to indoor air quality long before we knew indoor radon could be a hazard. I also commend them for today's hearing and for the one they held last October. This Science Subcommittee has been very responsive and willing to help address a very complicated issue and I commend my colleagues and all the members of the Subcommittee for their efforts.

Mr. SCHEUER. Congressperson Claudine Schneider, ranking minority member of the subcommittee. A very hardworking one at that.

Miss SCHNEIDER. Now, dear witnesses, I would like to share with you the fact that you all very much under the gun insofar as this program is concerned. I am sure that each of you followed the subcommittee markup of the indoor air pollution bill, and the full committee markup. The entire bill is now a part of Superfund legislation.

You may be aware that there was an effort to eliminate the funding for this program. The reason being is because my colleagues were not aware that this potentially is one of the most serious environmental threats that we are having to deal with. We are going to have to fight, those of us who are advocates of this program, tooth and nail to assure that the funding does come through. In order to do that we have to have a number of specifics that we can point to and say X, Y, and Z has been accomplished.

Now, Dr. DeLisi, you had mentioned that the Department of Energy has been in the business of researching indoor air for 10 years. What do we have after 10 years? You know, taxpayers and members of this committee like to see results. Could you give me some specifics as to what we have after your 10 years of research?

Dr. DELISI. Yes; the program in health effects of radon has not until the past couple of years been well focused. It has been at a very low level. And now I can say a few things about what has been accomplished, especially in the area of research which would lead to the development of the best mitigation measures. For example, it was previously thought that infiltration and ventilation, that is to say, the source of entry of radon into a home and ventilation of the home were uncoupled, they weren't related. But in fact, they are related, so that in fact ventilation is not—may, in fact, lead to a greater infiltration of radon into the home. So one has to be careful about that type of thing.

We've been able to identify the type of—the particular types of cells that are at risk in the lung. Now you may wonder why that's important. It turns out that the distribution of sizes of radon particles in a home is very complex. There are some very, very fine, small particles. There are some larger particles. The larger particles predominate and they lodge in the trachea. The small particles tend to interact with the more sensitive cells in the lung.

Now that has some implications for electrostatic precipitation methods. Because what happens with those methods is you remove a lot of the particles but you also generate more small particles, so that method may not be quite as effective as we would like to think.

We've also made progress at characterizing—it's not just the concentration of radon in the ground, but it's the properties of the soil. The moisture in the soil, other characteristics—the pH of the soil, which determine whether in fact—how much radon gas is released and gets into the home. Now that has implications for where you would build a home.

We've also started more recently with additions to our radon program an epidemiology study in the Reading prong and eastern Pennsylvania which would help us to better define, in fact, what the risks are at low levels of radiation, which we are all concerned with; that is, in the 4 to 20 picocurie per liter range.

Mr. Millhone, who is here from the Office of Conservation and Renewable Energy, may wish to add a few more pieces of information. And if you like, we can describe—we can submit for the record a list of specific accomplishments that are perhaps a bit more technical and it would take more time.

Miss **Schneider**. Yes; I would ask the chairman that we be able to submit that to the record.

[Copy of requested material follows:]

DOE Accomplishments Related to Indoor Radon

The Department of Energy and its predecessor agencies have conducted radon-related research since the 1950's. Early accomplishments included the development of the scintillation cell technique for radon measurement, study of radon exposures and airborne particle properties in uranium mines and mills, and investigation of indoor radon exposures and mitigation techniques contaminated with mill tailings (e.g., Grand Junction, Colorado). Studies of indoor radon not associated with industrial activities commenced in the mid-1970's.

Both the basic and applied research upon which current indoor radon exposure and health risks estimates are based has been sponsored by the Office of Health and Environmental Research (OHER) within the Office of Energy Research (ER). Complimentary radon research has also been sponsored by the Office of Conservation and Renewable Energy (CE). Several other offices within DOE have been involved in indoor radon activities. The Office of Nuclear Energy has responsibility for the remedial action programs associated with industrial activities. Indoor Air Quality information handbooks for consumers have been published by both the Bonneville Power Administration (BPA) and the Office of Environment, Safety and Health. The

BPA is conducting an extensive regional monitoring program in the Northwest, and has pioneered in field studies of mitigation techniques. Recent accomplishments of the DOE research efforts include the following:

Research/house studies sponsored by OHER and CE at the Lawrence Brrkeley Laboratory (LBL) and New York University (NYU) demonstrate significance of surrounding soil, as opposed to ground water and building materials, as the primary source of indoor radon and the importance of pressure-driven convective flow as a transport mechanism. Radon entry by diffusion is secondary. This has implications for mitigation strategies as well as for building technology.

The charcoal canister technique for radon measurement was successfully field-tested by the OHER's Environmental Measurements Laboratory (EML) and the University of Pittsburgh, and has become a widely-used commercial technique. These measurements are accurate within a range of 20% at normal indoor levels.

In 1983 LBL scientists analyzed various data sets and developed a preliminary National distribution of radon exposures. A more recent survey of approximately 500 houses of physics professors across the U.S. conducted by the University of Pittsburgh yielded a similar frequency distribution. This agreement provides confidence in the estimated National frequency distribution of radon exposures.

The OHER's EML participated in the Organization for Economic Coordination and Development (OECD)-sponsored quality assurance studies as one of four worldwide standard reference laboratories; conducted several international research laboratory intercomparison studies; and collaborated with EPA in developing U.S. radon measurement and quality assurance protocols.

Experiments with lung casts at New York Univrsity are providing more reliable data on the pattern of radon daughter deposition in the upper respiratory tract. This has significance for understanding the health effects of radon exposure.

A major epidemiological study sponsored by the OHER of lung cancer induced by environmental radon exposure has been initiated in the Reading Prong area in eastern Pennsylvania. This study is the first in the US. to study residential rather than occupational radon exposures.

With DOE funding, the BL and the Oak Ridge National Laboratory (ORNL) initiated collaboration with the EPA on diagnosis of causes of high radon exposures and development of criteria for choosing among mitigation technique options in individual houses in New Jersey and Pensylvania.

Research devoted to investigating the transport of radon through soils concluded that radon availability in soil appears to be greatest for moist soils. In addition, it was determined that convective flows dominate in coarse textured soils; diffusive transport in fine soils. This has implications for mitigation strategies and building technology.

Data tapes were processed from the National Airborne Radiometric Reconnaissance (NARR) survey. These data are used to provide three-dimensional surface plots of soil radium concentrations. This has implications for siting of buildings as well as attempts to identify high radon areas.

Tracer gas injection into soil was demonstrated to be an effective research tool for mapping radon transport through soils and radon entry into homes. This has implication for mitigation strategies and building technology.

DOE is working with EPA to develop a quality assurance program for commercially available track-etch and activated carbon detectors. In this program, which has thus far been very successful, private sector firms that fulfill defined measurement criteria are eligible for an EPA list of approved companies. The criteria testing has been held at DOE, as have more formal intercomparisons of research laboratory capabilities.

In existing structures, the radon situation is very complex. In the past three years it has been learned that both entry and ventilation rates depend on some common variables, such as pressure differentials across the building shell and are thus partially coupled. It is very likely, therefore, that radon entry in existing homes is strongly house dependent.

In new structures, the goal is to reduce radon entry rates. This can be done by appropriate and relatively inexpensive modifications of building construction practices, particularly with respect to the foundation. These modifications have yet to be fully defined but many are now being field tested by EPA and DOE. This will be an important contribution in solving the indoor radon problem.

Currently available information on specific indoor air quality topics has been synthesized by DOE into information handbooks for use by state and local public health officials, policy analysts, architects, engineers, homeowners and consumers. Handbooks on combustion sources and radon have been completed; a handbook addressing the impact of building system characteristics on indoor air quality will be completed in FY 1987.

Rodents have developed lung cancer at radon exposures as low as 20 WLM. Animal experiments also show a two to four-fold increase in lung cancer incidence per unit of exposure when exposure rates are decreased from 500–50 WLM/week. As yet, there is no explanation for this exposure rate effect. In any case, the applicability of the results of such animal studies to humans is uncertain.

DOE radiation effects research has been fundamental to the development of national and international occupational radiation exposure standards such as those recommended by the International Commission on Radiological Protection (ICRP) and the National Council on Radiation Protection and Measurements (NCRP). Information from these programs is also being used by the National Academy of Sciences (NAS) and the Biological Effects of Ionizing Radiation (BEIR IV) Committee in their development of a "best" estimate of radon risk.

DOE/CE and the BPA have developed, tested, and evaluated the effectiveness of energy efficient mitigation strategies to reduce indoor radon levels. These include subsurface ventilation, basement pressurization, air-to-air heat exchangers, and sealing holes and cracks.

The BPA has to date performed radon measurements in 17,000 homes, approximately 3% of which have radon concentrations above 5 pCi/1, the agency action level. These measurements indicate that geographical location is more predictive of radon concentrations in a building than its air leakage rates. They also indicate that the presence and concentration of indoor air pollutants is the major determination of indoor air quality, with ventilation having a lesser but still important effect. Therefore, steps to conserve energy which reduce ventilation, can aggravate a preexisting indoor air quality problem, but do not cause that problem.

Mr. MILLHONE. I'd like to summarize briefly the things that I think we have learned that have already been commented on to some extent. In the mid 1970's, we didn't really know radon was a problem, so we have learned that radon is a problem. One of the ways that we were able to determine that was work that developed the low cost test procedure.

We have also learned that the entry of radon is primarily pressure driven; that is, it's because of the relatively—a little less pressure in the basement of a house than outside, and that pressure drives the radon to enter the house, rather than a natural diffusion of radon gas.

We also know that the primary source of radon is soil gas, rather than water or the residue from some type of mining operation. We have also learned a great deal about how to mitigate the effects of radon through either sealing or caulking the basement or for the use of air, the air heat exchangers, or the use of positive ventilation outside of the house or the use of—so that the method of mitigation has also been an area of major progress.

The areas where more work is done I think is in more clearly understanding the health effects, the epidemiological studies, and then the location of radon in different parts of the country.

So we have made definite progress. There are still things that need to be done, and that's true of other pollutants as well.

Miss SCHNEIDER. OK. Thank you, Mr. Chairman. I just wanted to throw in a refinement of my question, and maybe my colleagues would be interested in hearing the answer to this.

As I mentioned from the outset, we are under the gun to produce; you are under the gun to produce. One of the things that I had recognized when we first introduced this bill in 1983 was that the Department of Energy and EPA had been essentially floundering around with research. There was not a cohesive game plan.

The legislation that has currently gone into Superfund has three specifications. First, it says to identify the sources of various pollutants typically found in the indoor air. Second, to identify what the health effects are believed to be caused by those respective pollutants. And third, to recommend mitigating measures. So it seems to me that if you are at this very moment following those three guidelines you will be a step ahead of the game by the time this legislation passes because the other thing that I added on to this was that you will have to report back to the Congress within 1 year and to give us intermittent reports so that we do not lose time and we do not lose money in solving the problem.

And I think that, you know, you gentlemen are certainly on the right path. But the fact that there has not been enough cohesion among the interagency designs of the game plan is unfortunate, and I think we need to hurry up and produce some substantial information.

And I would like to be sure that if any one of us were to call your offices this afternoon and say, we have some constituents that believe that they have some problems with indoor air pollution, would you be able to send us something now that is already packaged that, No. 1, would provide a diagnosis and, No. 2, that would give us the directions as to what mitigating measures can be taken immediately?

Thank you, Mr. Chairman.

Mr. **Scheuer**. I would like to know would you have that information, Mr. Ehreth.

Mr. **Ehreth**. I have some of that information summarized that I had planned, as I indicated before, to submit for the record, and it is generally scoped. But we can provide additional information, more details about those specific things.

Mr. **Scheuer**. In other words, you have a pamphlet or memo or what not——

Mr. **Ehreth**. Oh, I'm sorry. Oh, I apologize.

Mr. **Scheuer** [continuing]. That we could give to constituents who are concerned——

Mr. **Ehreth**. Yes.

Mr. **Scheuer** [continuing]. That they may have a radon problem in their homes.

Mr. **Ehreth**. Yes, sir; we definitely have that in radon. Let Mr. Meyers address that.

Mr. **Meyers**. We are talking about two things here: indoor air, in general—

Mr. **Ehreth**. Yes, that's right.

Mr. **Meyers** [continuing]. Of which radon is a part, and——

Miss **Schneider**. And I want the big picture of indoor air.

Mr. **Meyers**. I understand that, but I'd like to just say a few words about the radon program because many of the things you are concerned about not having happened in indoor air are well underway in the radon program. We have the documents you're talking about. We have pamphlets that we can distribute to people that describe the nature of the problem and what you can do about it. We have, also, in the various categories I've mentioned earlier developed standardized measurement techniques so that when people take measurements they do it consistently and in the right way. We have developed a quality assurance program so that people who are out there offering their services to citizens have a way to know that—the citizen would have a way to know that they are not being taken.

These programs are in place now. We have a national survey for radon in the design stages now, and we expect to initiate that survey next year. So what I'm saying is for radon we do have a program in place in accordance with a plan that has been approved by the Administrator.

Mr. **Ehreth**. And to continue just briefly, for some of the other pollutants related to the indoor air problem, we would not have been able to do that, we would not have been in a position to do that had we not have been able to accomplish some of the things we did initially with regard to instrumentation development, for instance. So we are just beginning to measure that.

The three points that you shared with us in that bill are, in fact, the three points that represent a more balanced program, and that is the more balanced approach that we are currently pursuing. So again, in response to that particular type of a message.

Miss **Schneider**. Great. Thank you.

Mr. **Scheuer**. Well, I'm going to ask you if you would be kind enough to send us a couple of dozen copies of whatever materials you have for distribution to members of this subcommittee and the full Science Committee.

And then I would ask staff to do a little scrutiny—perhaps you can help us—identifying the portions of the country that have the greatest radon problem. And at least as to radon we could then alert Members of Congress who represent those areas that there is something available for them to be providing their own constituents.

Mr. **Ehreth**. We would be glad to do that, sir.

Mr. **Scheuer**. OK. We appreciate that very much.

OK. Congressman Bart Gordon of Tennessee.

Mr. **Gordon**. Mr. Meyers, as you know, I recently wrote EPA Administrator Thomas encouraging the EPA to go forward providing Tennessee with 3,000 radon detector units and to conduct a survey, provide expertise in evaluating the results, and recommend a course of action. And I am pleased, after speaking with you earlier, that you are going forward with that, and I want to thank you and EPA for that.

Let me ask that if Tennessee or another State is determined to be a hot spot, or an area in that State is a hot spot, what kind of expertise and financial aid can EPA or any other governmental agency offer to the States to try to remedy the matter?

Mr. **Meyers**. At the moment the program is designed to identify the problem and to describe what you can do about it. There is no provision in the Federal program to provide financial assistance to the homeowner so that he can correct the problem.

Now, I should—too bad Congressman Yatron has left, because Pennsylvania has probably a fairly ideal program, where we helped initially to find out the nature of the problem. They picked about 2,000 or 3,000 homes. They then surveyed some 20,000 homes, discovered elevated levels in about 60 percent of them. They then set up a low interest loan program where, I believe, if your income was below $33,000, you got a 3-percent loan, and an 8-percent loan above that. They used our brochure to—after the measurements were given to the citizens, the brochure we put out said if your level is at such-and-such level, then you ought to be thinking about remedial action; turn to the State, the low interest loan program.

I think that integrated program of the Federal Government doing what it does best and the States providing assistance is a model way to do it.

Mr. GORDON. So as far as how deep you're going to get into this, basically you're going to come down and help the—I guess by providing expertise, help the State to have the survey, and then to really educate the State staffs to go forward. You're not going to be, or the Federal Government, EPA, is not going to be going into the individual homes and counseling; you're just going to try to get a—

Mr. MEYERS. Not on a regular basis. We will do some of that. One portion of the program that I should have mentioned earlier that has been very successful has been a training program where in the past 2 or 3 months we have run diagnostic training programs mostly in the Reading Prong. I think we did about 20 of those, and we will continue them in other parts of the country this next fiscal year.

Mr. GORDON. Let me ask you, if you have a State individual who is working in the health field who doesn't have a great deal of expertise in this area, walks into you, what kind of time and expense are going to be involved in trying to get a State up to speed in being able to go out and help others?

Mr. MEYERS. All the States do have radiation departments of one kind or another, so they generally have the kind of technically trained people that would be the kind of people that would mount these kinds of programs.

Mr. GORDON. But as we I think have established already, it is pretty vague what some of the hazards are, and there isn't that information. Much of it has come about from looking at miners and determining whether, you know, trying to extrapolate that over to the radon situation in a home. So it is still pretty questionable. So if you're not really aware of what's going on, then there can't be too much expertise on the State level, either.

Mr. MEYERS. Well, we're not expecting any of the State people to do the epidemiological studies. A number of Federal agencies are involved in that the Department of Energy is running one. The National Cancer Institute is running another one. So, you know, we're aware of the limitations of the miners studies, but we're still convinced that radon represents a health threat and it needs to be addressed even though we're still refining the health estimates. So the people that I'm talking about in the State programs are familiar with the kinds of instrumentation that it takes to measure radon. They can measure radon. They can find out what the levels are, and we will then provide the information that says these are the risks associated with it.

Mr. GORDON. What would you see as a—I'm sorry, did you——

Mr. MEYERS. No, no.

Miss SCHNEIDER. No, I wanted to ask my colleague if he would yield for a moment.

Mr. GORDON. Certainly.

Miss SCHNEIDER. I'm curious too. I think it's important for us to make the distinction, if we are assuming that, say, for example, one community in your State believes that they have a problem with radon pollution, the inclination would be to call their State Department of Environmental Management or the regional EPA office.

But I think that what you gentlemen need to point out is that there are private sector options. That if, for example, I wanted to test the radon levels in my home right now, I could pick up the phone, call a toll-free number, I believe, and find out where I can get a canister, where I would pay $50 for this canister, put it in my home for *x* period of time, send the canister in, and they would do the analysis of it to let me know what levels of radon I'm being exposed to in my home.

So I can just imagine after, if an article like the one in the Washington Post appeared, to have all the State of Rhode Island calling the State environmental office and saying we want the State to come in and test our office building or test our home. You don't have to take that route. As a matter of fact, I think that the preferred route would be to take the self-initiative and go to the private sector and get one of these canisters yourself.

Mr. GORDON. I think a lot of individuals will. But also I think there is a need, rather than to—and it is not haphazard to go here and there, and for the individual to know, but I think it's also a need to determine regions. Everybody will not have the opportunity to read the Post or to see that information. Some of the more informed may very well do that, and can afford $50. Many others won't be able to either know about or afford the $50. And I think it's important that we be able to determine the hot spot regions so that there can be an affirmative and aggressive action to go into those areas.

Mr. EHRETH. Mr. Gordon, may I add something to Mr. Meyers' statement? We are taking two approaches in the risk mitigation aspect of the radon problem. We have in conjunction with the Office of Radiation Programs developed a little pamphlet, which you'll see copies of, for the homeowners; for the average citizen, to help him understand what he might or she might do with regard to risk mitigation.

Second, we've prepared a manual for the design community, or for the builder's community. And through setting up programs at State universities and what not, hoping, you know, through educational processes of that nature to educate the builders of homes and of the buildings as to how to design a radon-proof structure, so to speak, and take the radon issue into characterization at the beginning of the program.

We have copies here for you on those particular things we haven't showed you.

Mr. GORDON. I have seen that. Beulah the buzzer has rung, but Chairman Scheuer was kind enough to give me one more question.

You know, you've got a couple of different areas to deal with. Obviously, I think we're dealing from the premise that radon is a hazard, and you're still trying to determine, you know, what level is that hazard and when do you become concerned and, you know, is a one-pack-of-cigarettes hazard or a four-pack-cigarette hazard, those sort of things.

The other thing that I think that you've got to determine on this national survey is, again, where are those hot spots, where do we need to take an aggressive action to go forward affirmatively and encourage people to do something. I know you're in the process of that.

Could you give me some estimation of a timeframe in trying to survey nationally, determining where the hot spots are, and determining where we need to do more than just read in the paper, where we need to get in and aggressively go in and try to address the problem and find remedies?

Mr. MEYERS. Yes. The national survey is being designed right now, and we expect that to be completed next year and to initiate the emplacement of canisters. Overall, we expect the entire program to be finished 3 years from about now, but there will be data that comes in intermittently as the program proceeds. It is not a case of waiting totally till the end of 3 years before we get any information. That would be the period of time when it would all be wrapped up and a report would be issued. But we would be getting information from various States on the high levels and from our own program on the average levels on a continuing basis.

And of course, if we identified any particular hot spots, we would so notify the State.

Mr. GORDON. And as you do this, you are going to come forward with recommendations that we need to implement legislatively to take care of the problems and determine remedies?

Mr. MEYERS. Well, right now we don't feel that legislation is needed in the sense of what the homeowner ought to do. We have already put out the brochures that explain what the levels are and what the risks are and what you ought to do about it. So the point now is to find out whether or not you have a high level that needs something to be done about it.

Mr. GORDON. I think the important thing here is that we began to take action to provide answers as to what to do about the problem before all the scientific debate ended as to how extensive the problem really was. I think that's what's important in this radon area.

Mr. SCHEUER. Congressman Bob Smith of New Hampshire. We've relocated you back to New Hampshire, Bob.

Mr. SMITH. I was going to say I do like Vermont. I know it's closer to New York. [Laughter.]

Thank you, Mr. Chairman.

In New Hampshire, of course, ground water is very critical, as it is in other States. How extensive is the radon problem in groundwater? Anyone who——

Mr. MEYERS. Nationally, water contributes about 15 percent to the total radon problem—nationally. Unfortunately, up in New England it is a lot higher. I'm not quite sure of the exact numbers, but it is much higher in Maine that I know of, for example.

Mr. SMITH. Does your survey intend to monitor or in any way measure the problem of ground water, radon in ground water or is it going to be exclusively to monitoring what's—the gas in the homes?

Mr. MEYERS. The survey that I have been talking about is designed to measure indoor radon only. I think there have been other surveys to measure radon in ground water. I think they were done a couple of years ago.

Mr. SMITH. Just as a for-your-information-type thing in the event that you haven't heard of it—maybe you have—in my congressional district in New Hampshire there was a trailer park which installed a charcoal filter to filter out the radon particles present in the park's water supply. In addition, of course, they also trapped other contaminants, whether it be uranium, or whatever, radium, thorium—whatever is in it. And after that filter was removed, after a certain period of time, it was classified as low-level radioactive waste, higher than the minimum State requirements—exceeding State requirements.

The question is, What does one do with that filter? And I guess, are you also, in addition to recommending mitigation methods, are you also looking at the disposal problems that may be related to this type of thing?

Mr. MEYERS. It so happens we are. The reason that filter was so hot is that it filtered out radium, and we are developing under a different part of our office a low-level waste regulation that does include what is called radium as a naturally occurring radioactive material, that is not covered under the Atomic Energy Act, and we are planning to put out this low-level waste regulation to govern the disposal of naturally occurring radioactive material as well, radium being the major component of that.

Mr. SMITH. I have no further questions at this time. I should be glad to yield to anybody else who may have another question, however.

Thank you.

Mr. SCHEUER. Can you tell us, and I guess this would be either Mr. Ehreth or Mr. Meyers, what is the basis for the risk estimates included in the chart contained in the "Citizen's Guide to Radon"?

Mr. EHRETH. They're essentially——

Mr. SCHEUER. These seem to be pretty mushy figures. Can you tell us where they come from, and how hard you think they are?

Mr. EHRETH. They are from the miners studies that you heard about. And I guess I wouldn't use the word "mushy."

Mr. SCHEUER. Well, what word would you use?

Mr. EHRETH. Well, they're very good figures in the sense of the data we have. We do know that of the studies that were done there were a statistically significant increase of lung cancers in these miners. Now the—I would use the word "uncertainty." And the uncertainty lies in the fact that at the time the miners were exposed there weren't measurements taken in the mines, so that what we had to construct, and the reason for the uncertainty, is the exposure that these people were faced with.

So the uncertainty that we have here is related not to the fact that there were a number of significant increases in lung cancer, but what the level of exposure that these miners had at the time. And various researchers have constructed, based upon analyses, what they thought it was, so you get a range of exposures and risks associated with those exposures because of that.

Mr. Scheuer. What I'm trying to get at is that given, or when we look at all these assumptions, isn't it correct to say that there are significant uncertainties connected with the projections about health effects, certainly in the home? How do we characterize these uncertainties, and how do we tell people how to cope with their uncertainties?

For example, your 0.02 working level. If we have 10 times the number of people at 0.04 that we have at 0.02, and we'd be spending vastly greater amounts of money, is it possible to tell people that they have an *x* risk at 0.2 and a *y* risk at 0.4? And if their home is easily susceptible to remediation efforts for a few hundred dollars, why, they should go ahead at 0.02; but if it would cost them thousands of dollars and it might be that 0.02 might be the equivalent of smoking a half a pack of cigarettes a day, they might want to wait to spend those many thousands of dollars until their home achieved the level of 0.04.

Shouldn't individuals be brought into the decisionmaking circle, so to speak, and informed as to exactly how significant these measurements are, how serious the risk is, how much of a reduction in risk they would make going from a 0.04 level to a 0.02 level, so that they could figure out whether it was worth the capital investment, if it was a major investment, to reduce the risk that distance?

Mr. Meyers. There is no doubt, Mr. Congressman, that there are uncertainties. The level at 0.02 was selected in large measure because it's a practical lower limit than one can get to. And obviously, if the limit were at 0.04, that would drop out a large number of houses. But even at 0.02, there is a substantial risk.

Now, while there is uncertainty, we feel that the extrapolation from the miners' data—and in many cases some of the levels we've seen in homes are equivalent to what you see in a mine—we have, indeed, factored in the fact that a home is not a mine in terms of occupancy rate, breathing rates. So the levels have been adjusted to reflect that a living room is not a mine.

But in terms of regulations that EPA routinely writes on other pollutants, we certainly know much more about radon than we do about many of these other pollutants, in large measure because the data we have are on human beings. So when you get down to an individual making a decision, as you go through our brochure you will note that these are recommendations. There is nothing in there that forces anybody to do anything. And we tried to stratify it in a way so that if there were a sense of urgency at the much higher levels, like one working level, we suggest, you know, you do something fairly quickly. But down at the 0.02, 0.04 level we are talking about in the next couple of years you ought to think about getting it fixed.

So we tried to factor in the uncertainty in the way the brochure was written.

Mr. Scheuer. I have a question that I'd like both Mr. Ehreth and somebody from DOE, Mr. DeLisi, to respond to. In March and April of 1986, despite opposition from the CIAQ and outside scientists, EPA entered into contracts to proceed with part of the survey design. At that time a fairly complete survey plan existed. Was that complete plan ever shared with other members of the CIAQ?

Mr. **Ehreth**. That plan, that request for proposal was canceled before it went on the street. It was initiated because at the time that it was initiated we were still thinking about doing the national field survey; and consequently, this was still back before we had the meetings with scientists and those people. We had been discussing that field survey with the CIAQ and in the field studies work group, yes, many times, Mr. Chairman, over the past several months before that period of time.

And you are correct, there was not unanimity in terms of support of that field survey. We felt at that time that we were responding to congressional interest as well as our initial peer review that we conducted back in the fall or late—early winter, I should say, of 1983–84. We felt very strongly that at that time we had received the direction to go for the national field survey. That RFP, as I said earlier, did not reach the streets; it was canceled. That was the field survey that I alluded to in my opening remarks that is now off the stove.

Mr. **Millhone**. I would like to answer that question, and perhaps a little broader question that gets involved in the GAO report and also the role of the CIAQ. As one of the cochairmen of the CIAQ——

Mr. **Scheuer**. I think we ought to just say for the record the CIAQ is the Committee on Indoor Air Quality.

Mr. **Millhone**. Committee on Indoor Air Quality. Yes.

As one of the cochairs, I'm concerned about the perceived disagreement that you've attributed to the CIAQ. I believe that the organization has made significant progress in coordinating Federal indoor air quality research activities and that the record of how that is done now is significantly better than it was before the CIAQ was organized.

I think that the multi-pollutant field study is an example of how that coordination has worked. Where there were legitimate disagreements about the methodology that would be used, the phasing, the funding and some of the approaches that were initially proposed, it received a vigorous critique by other participants in the CIAQ, and as a result, EPA has made changes in the approach that it's taking. And now I think that the direction that the EPA is taking in this area is one that we enthusiastically support.

I think that because of the nature of the indoor air quality issue different agencies such as EPA, such as Health and Human Services, such as DOE, such as CPSC—all have significant interests that bear in this field and that, therefore, the interagency approach is the proper approach. I think that the Congress is very appropriately holding our feet to the fire from time to time to make certain that the coordination is effective.

Mr. **Scheuer**. Well, let me put your feet to the fire a little bit further. How long did it take you to make that change?

Mr. **Millhone**. It was undertaken I think over the course of about a 6-month period from when the issue first was faced and then the reworking of——

Mr. **Scheuer**. We have memos that go back 2 years discussing this matter.

Mr. **Millhone**. Yes. As I said, again, at the outset, when we sent over our first plan, it was in 1985—the first plan on the interagency committee's activities. One of the six tasks identified in that plan was national field surveys, or field studies. And so we had been discussing it many, many times. Initially it was intended to be, it is my recollection at least, is to focus on——

Mr. SCHEUER. Was it roughly over a 2-year period that all these discussions went on?

Mr. MILLHONE. Discussions of many things were over a year or two-period, yes, sir.

Mr. SCHEUER. What else did you accomplish during that 2-year period?

Mr. MILLHONE. Well, as I said earlier, we—during that period of time while we were planning for the field study, we still had the instrumentation to develop and field test, and things of that nature; the survey design protocols, and many other things.

Mr. SCHEUER. OK.

Mr. MILLHONE. It's impossible to jump right into a national field study, just as the radon people would say, without having the instrumentation and the knowledge of how to put together an effective protocol for such a study. So this was a discussion issue over the years, and it took many shapes over those, over that 1- to 2-year process. It was finally resolved after we sat down and after I had convened the cochairs and a group of scientists, and we sat down and we decided how to resolve it.

Mr. SCHEUER. It is my understanding that you spent substantially 2 years in discussion as to the kind of survey that you would have, and now you have agreed to postpone the survey.

Mr. MILLHONE. That's right. One of our subgroups is called the field studies group, and it's their purpose in the CIAQ activities to address field studies which are of a survey type. And they would review these types of things for us.

Mr. SCHEUER. All right. We really don't get the impression that there's a concentrated focus, organized, disciplined effort to produce hands-on information that the Congress can use and that the people can use to—in the workplace and in the homeplace. Something is lacking. And I think the diffusion of the discussion of the last few minutes, the mushiness of the answers, indicates that something is wrong. Something is rotten in Denmark.

Mr. MILLHONE. Well, simply stated, to conduct that field survey, we had to also develop the methodology and instrumentation necessary to do it. And in doing that we had to decide what kind of methodology was important. So the discussions did ensue.

Mr. SCHEUER. It seems to us that you have discussion ad infinitum. The discussion seems to be the beginning and the middle and the end. Discussion for the purpose of more discussion for the purpose of more discussion without any focused end in view, in sight. And I'm not an expert. I haven't spent full time on this. I don't begin to know as much about it as I should properly to conduct this hearing. But I've got a feeling that you lack direction, you lack focus, you lack ability to design a research program that has a beginning, a middle, and an end and that will produce some results that the Congress and the people can use. Now that's a feeling I have, and I think other members of the committee have the same feeling.

Mr. MILLHONE. Well, OK. Thank you very much for your testimony. We'll move ahead to the next panel.

Mr. SCHEUER. All right. Thank you for your patience.

We have a very distinguished panel with us here this afternoon.

Dr. Jan Stolwijk, in addition to being a distinguished and world renowned expert on indoor air pollution, is the chairman of the EPA Science Advisory Board reviewing EPA's indoor research—indoor air pollution research plans, and therefore he brings a special expertise with him here today. And perhaps he can tell us that we don't need to feel as frustrated as we do feel.

Richard Zimmer is chairman of the National Council for Clean Indoor Air. This is a newly formed coalition of public interest and private sector groups all of whom have a special interest in indoor air pollution. So at last we now have an entity that can represent the interests of all of the groups, both in the public and private sector, involved in indoor air pollution. And we will hear briefly from three of these groups.

All right, we'll start off with Dr. Jan Stolwijk, chairman of the Department of Epidemiology and Public Health of Yale University School of Medicine, and also Chairman of the EPA Science Advisory Board, which reviews EPA's indoor air pollution research plans.

Dr. Stolwijk.

If you would all take approximately 5 minutes, your testimony will be printed in full at the point in the record at which you testify. We hope that you will just address us quite informally, and chat with us, and then I'm sure we'll have some questions for you.

Dr. Stolwijk.

STATEMENTS OF DR. JAN STOLWIJK, CHAIRMAN, DEPARTMENT OF EPIDEMIOLOGY AND PUBLIC HEALTH, YALE UNIVERSITY SCHOOL OF MEDICINE; RICHARD ZIMMER, CHAIRMAN, NATIONAL COUNCIL FOR CLEAN INDOOR AIR, ACCOMPANIED BY KEN SEXTON, DIRECTOR OF RESEARCH, HEALTH EFFECTS INSTITUTE; AND A. JUDSON WELLS, VICE CHAIRMAN, NATIONAL COUNCIL FOR CLEAN INDOOR AIR

Dr. STOLWIJK. It is a pleasure for me to have the opportunity to testify before your committee, which is I think singularly well informed in the area. And as someone who has devoted energy to this area, I am highly appreciative of the committee's efforts which I think are highly appropriate and serve the public well.

As we've heard from earlier testimony, we're concerned with a very large number of diverse spaces, from every room in every residence to every office and every classroom or meeting room in the country, when we're discussing indoor air quality. The quality of the outdoor air, which we rely on to dilute whatever it is in the indoor environment that we don't wish to have, has substantially improved in the last 15 years or so. And I think that the EPA and the Congress is to be commended for the very large improvements that have taken place.

We're now tackling indoor sources because it's coming to be recognized that, in fact, the indoor environment may be less healthful than we have managed to make the outdoor environment at this point. The problem that we're faced with is quite complex and very interdependent. The sources are important, the ventilation rate is important, and knowing what the effects are—all determine the optimum point of attack and the optimum strategy for attack.

The improvements that will result will be the result of choices made by householders and choices made by building managers based on the clearest and the most usable information that we can provide. Indoor air quality is going to be provided with the people, whereas outdoor air quality could be provided for the people without their active participation.

In order to deal—the indoor air quality problem is inherently much more complex than the outdoor one. It is also more difficult. It will resist solutions for a longer period of time. Progress will be slower. Nevertheless, the necessity for the comprehensive insights that are needed requires a well-designed and well-managed research program which keeps track simultaneously of all the factors that are involved, calling on the contributions of the different disciplines as they are appropriate.

In the review that was made of the EPA program by the Science Advisory Board panel there was, I think, unanimous agreement about the quality of the scientific effort that was devoted to the problem, but there was also a recognition that the kinds of questions that were trying to be answered were, perhaps, not the most relevant questions that could be asked.

I think that the panel will recommend that the EPA engage in the setting up of some sort of internal accounting which can be shared with all concerned; that makes a statement about the range of exposures that occurs for every pollutant; that makes a statement about the estimates of health effects that are associated with these and, consequently, the total public health impact; and that makes a statement about what kinds of avenues are available for mitigation.

Because it is a multidisciplinary problem, each discipline will have to supply those elements that it can, and the unification will take place in the fact that they will be related to one another and that they will be related to existing studies and prior studies. So that based on that kind of diagram it should be possible for all of us to see where progress is being made; where, in fact, the holes exist; and how best progress can be made.

It will still not really be possible to make a universally acceptable rank order of these problems. Cancer and head colds do not compare very easily, even if you have the numbers. The things that we're concerned about in some kind of order, but this has to be a personal order, are those of passive carcinogen smoke, radon, NO_2, microbial contaminants, and organic contaminants that are brought into the structure or that off-gas from the structure. This is a rank order that perhaps might reflect personal prejudice, but it is important that in an indoor air program the microbial contaminants, for instance, not be ignored, which is likely to occur unless a comprehensive list of indoor air pollutants is actually made up and justified. It is very likely that in the current context microbial contaminants, which have become an unpopular item in the research agenda of the Nation, they are likely to be ignored, and I don't believe they should be. They still represent a very important part of maintaining a healthful internal environment.

Until a systematic and quantitative and written assessment of the distribution of sources and our understandings of them, the distribution of exposures and our understandings of them, and the distribution of health outcomes, is known, we will not have a way of actually rewarding the work that needs to be done and discouraging the work that doesn't need to be done anymore. There is no yardstick at the moment that helps everyone, including you, decide what is the appropriate pathway to follow next.

As was mentioned, there is a development within EPA that the Air Office is going to develop a policy statement of some sort that would relate to indoor air quality. I would hope that such a statement would actually come from the Administrator himself as to what the task is that EPA sets for itself. I think that a thorough understanding and a thorough development of an indoor air program may also be helpful in reinterpreting and evaluating the efforts that are continuing in the outdoor program.

The outdoor ambient air quality standards clearly must be seen in a perspective that relates them to the overall exposure that the people have to these values and to the exposure that people have to these contaminants in the outdoor and in the indoor environment simultaneously, so that there is a coherent picture that allows you to make judgments as to where the best and the most feasible gains can be made.

I think that it was the feeling from—by the Science Advisory Board panel that the EPA staff that presented its material was eager, committed, and competent, and that with proper organization and proper focus that we can look forward to considerable improvements in the effectiveness of that program.

I would be happy to answer questions on this or on the written testimony that I have submitted. Thank you.

Mr. SCHEUER. Thank you very much, Dr. Stolwijk.

[The prepared statement of Dr. Stolwijk follows:]

PREPARED STATEMENT OF JAN A.J. STOLWIJK, DEPARTMENT OF EPIDEMIOLOGY AND PUBLIC HEALTH, YALE UNIVERSITY SCHOOL OF MEDICINE

Mr. Chairman, and Members of the Committee, I am Jan A.J. Stolwijk, Chairman of the Department of Epidemiology and Public Health of the Yale University School of Medicine and I wish to thank you for the opportunity to provide testimony at this hearing. This Committee has demonstrated great insight into the problem of indoor air quality. Especially considering the range of your responsibilities, I am gratified, as someone with a sustained interest in this area, to have an opportunity to appear before you.

The quality of the indoor air we breathe is of much greater primary importance than the quality of the outdoor air which is so carefully being protected by the current regulations of the Clean Air Act and its amendments. The simple and compelling reason behind that statement is that we spend 80 to 95% or more of our time indoors. Ultimately the quality of the indoor air environment must depend on the outdoor air quality because we rely on outdoor air to dilute the contaminants being introduced into the indoor environment. Attention to the indoor environment in no way should detract from the very successful efforts currently in effect to protect the outdoor ambient air quality, a program which can proudly point out the substantial improvements in the nation's ambient air quality which EPA has achieved since 1970.

Your Committee is well aware that the strategies which will be necessary or effective to provide the citizens of this country with healthful air in their residences and in all buildings with public access are very different from the strategies which served us so well in cleaning up the ambient air outdoors. There is likely to be much less in the form of regulation, and much more in the form of providing information to householders and to managers and owners of buildings which will help them carry out actions which will promote more healthful air inside buildings. The difference extends also to the research programs and strategies which will be necessary to provide the necessary information to such users. In fact your Committee, Mr. Chairman, appears to have seen this earlier than the Environmental Protection Agency or the Administration.

The European Regional Office of the World Health Organization recognized the special needs for Indoor Air Quality Research and has published over the last six years a number of working papers for its member governments; the latest one, which has become available only in the last few weeks, specifically addresses research needs and mechanisms to arrive at priorities for such research. This document is entitled: "Indoor air quality research" it is No. 103 in the Series "EURO Reports and Studies" (available from: WHO Publications Center USA, Albany, NY 12210) and will be made available to your staff.

The Environmental Protection Agency should have clearer legislative direction to specifically take responsibility for providing help to the U.S. population for achieving a higher indoor air quality. The lack of specific direction has made it difficult to impossible to formulate and state a clear policy with clear objectives against which its authorization and appropriations as well as its progress can be measured. Without such a clearly stated policy and objectives it is difficult for the scientists and engineers within EPA to organize and focus their considerable skills and interest in this important area in ways which help the users to recognize and solve their indoor air quality problems.

One of the most important ways in which indoor air quality problems develop and get solved is through the ventilation systems which serve buildings. The American Society of Heating, Ventilating and Refrigerating Engineers is a professional organization which serves the country in a number of important ways. One of these important contributions is the development of consensus standards governing all aspects of heating, ventilation and air conditioning. This Society is currently revising its Ventilation Standard, which is the only standard of this type we have in the U.S.A. and which gets incorporated in building codes. This Society is carrying out a revision and update of its ventilation standard, and it can find precious little in the way of governmental research activity which supports it in this effort.

There may be merit in professional organizations developing consensus standards without direct governmental participation, but such organizations cannot do the research necessary to support such standards and their meritorious efforts currently suffer from inadequacies in the data upon which improvements in the ventilation standard must be based.

In a recent review of the Indoor Air Quality Research of the Environmental Protection Agency which was conducted by its Science Advisory Board it was clear that a major limitation imposed on the effectiveness with which EPA can serve this area is the lack of a clear policy and clear objectives within the Agency. Most who read the letter and the intent of the Clean Air Act and its Amendments, and who are aware of hearings such as this one, can see the absolute necessity of EPA taking responsibility for Indoor Air Quality. It appears that there are voices in this and preceding Administrations who instruct the Agency otherwise. I hope that Congressional efforts to clarify its instructions will be successful.

One of the indoor air quality concerns which recently has become more widely noted is that associated with exposure to radon daughters. It is known from surveys in different regions of this country, as well as in a number of other countries, that there is a very wide range of radon daughter exposures which exists in residential environments. We will soon have better data about this distribution so that we know how much of the U.S. public is exposed to how much in the way of radon daughters. These studies are being carried out in a number of regions of the country. There are data from mortality in uranium miners as well as animal data which can be used to estimate the number of lung cancer deaths in the U.S. which might be ascribed to radon, and which could be prevented if radon exposures were eliminated or reduced; numbers of 5,000 to 20,000 lung cancer deaths nationwide which might be attributed to radon have been mentioned.

In order to retain a perspective it is well to remember that in 1930 lung cancer mortality among women was 1 per 100,000 per year. (At that time this mortality was 5 per 100,000 per year in males). It is reasonable to accept that radon daughter exposures have not changed materially since 1900 or 1930, and this then places a maximum on the contribution of radon daughters to lung cancer of perhaps 0.5 per 100,000 people per year. Translating this to the U.S. population would make the current lung cancer mortality due to non-occupational radon daughter exposure have to be something less than 1,100 per year. By comparison current lung cancer mortality for men is of the order of 70–80 per 100,000 per year, and for women 25–30 per 100,000 per year, for a total of 85,000 men and 31,000 women each year. If this comparison is justified it indicates that the projections based on uranium miners are inappropriately applied to the general population and we may then not be quite ready to justify a firm radon mitigation policy on anything but the highest levels encountered.

It can also be seen from the above example that in a complex problem such as indoor air quality an almost infinite number of kinds of studies can be visualized, but only relatively few are simultaneously relevant, feasible and urgently needed.

Within the Federal Government we have an enviable array of talent, skills and experience in a number of agencies which are coordinated through the Committee on Indoor Air Quality (CIAQ). Even though EPA is clearly a lead Agency in the overall problem, several other Agencies have important experience and knowledge such as DOE in radon and ventilation, CPSC in formaldehyde and consumer products, HHS in tobacco smoking, etc. Policy statements, objectives and strategies within these agencies and between agencies will become more effective with a clearer directive from the Congress to EPA and this witness urges such directives.

Mr. **Scheuer**. Now we'll hear from Richard Zimmer, chairman of the National Council for Clean Indoor Air.

Mr. Zimmer.

Mr. **Zimmer**. Mr. Chairman, members of the committee: on behalf of the National Council for Clean Air I thank you for the opportunity to testify.

I beg your pardon?

Mr. **Scheuer**. Clean Indoor Air.

Mr. **Zimmer**. Clean Indoor Air, yes, sir. Thank you.

I thank you for the opportunity to testify regarding this important and growing issue.

I'm Richard Zimmer, chairman of the council, and a State assemblyman from the Reading Prong area of New Jersey. I'll be speaking from a State legislator's point of view, while my two colleagues will provide some of the finest technical expertise available in the country today. They are Dr. A. Judson Wells, who was with the Du Pont Co. for 37 years as a scientist and senior executive and who has since his retirement been very active in the analysis for the passive smoking issue; and Dr. Ken Sexton, now director of scientific review for the Health Effects Institute in Cambridge, MA, and formerly director of the Indoor Quality Program for the State of California.

We have helped to establish the National Council for Clean Indoor Air because, despite a growing awareness in the science and health fields, indoor air quality is still unknown to much of the general public and to many of your and my colleagues in public office. The council will be bridging that gap, making scientific and medical research available to public policy officials in an understandable and readily available form.

Having been the principal assembly sponsor of the New Jersey radon remediation law and a recently enacted law to certify persons who test for radon and who mitigate radon contamination, I can say that a body of experts such as this council would have been an invaluable asset as we were struggling with this new public health issue.

As it should be with all new issues, there is an important debate going on regarding the proper role of the Federal Government. There is agreement, however, regarding issues of basic scientific and medical research. Clearly the Federal Government has a role to play in determining the incidence of the problem, the health effects and the health standards for radon exposure. Beyond these basics there is a need for an intensive examination of the many activities which make up our public policy: assessment techniques, remedial technologies, examination of the characteristics of radon, financing of remedial actions, public education, and communication between private and public, and State and local, Federal and international agencies. They are all unsettled issues of responsibility.

Radon, in particular, and indoor air quality as a whole are relatively new environmental health issues. But in addition to thrusting in front of us serious matters of public health, these issues also present us with an interesting opportunity. Indoor air quality issues carry little of the baggage associated with other health and environmental issues. There is an opportunity to start at the beginning and develop a sound marriage of science and public policy, and to utilize in their proper roles Federal and State action, private industry, and individual initiative.

If we are to achieve this successful blend, indoor air quality must not be allowed to float between agencies. Lead responsibility must clearly be assigned and a long-term commitment to serious budgetary resources must be made.

Management officials at EPA and other Federal agencies and the policy officials at OMB must understand the congressional commitment to this important public health issue. In the GAO report on radon to the Pennsylvania congressional delegation the GAO restated its previous recommendation that EPA be designated the lead agency on indoor air quality issues, and added to its original recommendation the specific recommendation that it be the lead agency for radon issues as well.

Among other things EPA should be responsible for assuring that radon research efforts do not overlap, that radon research is being conducted on the most significant issues, that the Federal Government is operating under and recommending consistent radon policies and guidance. We feel that such a policy statement by this committee would be of great benefit to EPA in creating an effective and efficient radon program.

We would also concur with the GAO finding that formal designation should not interfere with the radon interagency groups which have recently formed. Further, the specific designation of responsibility will make your oversight duties more manageable, accountability more clear, and the desired results more likely.

It is interesting to note that EPA's Science Advisory Board subcommittee has made similar recommendations to EPA regarding the Indoor Air Quality Program as a whole. I won't go into any more program specifics, as I do not want to repeat any of Ken Sexton's overview of indoor air and his discussion of the need for a systematic approach to these many issues, radon included.

As I mentioned in the opening, New Jersey has recently enacted a radon remediation law and a certification program. I won't go into specifics at this time, but would be pleased to answer any questions you may have. I hope we'll have the opportunity to discuss some of the things we learned, what the public reaction has been to this problem, and what the Federal Government provided and what they didn't.

At this time I would like to give Dr. Wells the opportunity to share with you some of his own research regarding the severity of the passive smoking risk.

[The prepared statement of Mr. Zimmer follows:]

TESTIMONY OF RICHARD A. ZIMMER, CHAIRMAN, NATIONAL COUNCIL FOR CLEAN INDOOR AIR

Mr. Chairman, ladies and gentlemen of the committee, on behalf of the National Council for Clean Indoor Air, I thank you for the opportunity to testify regarding this important and growing issue.

I am Richard Zimmer, chairman of the council and the State assemblyman from the Reading Prong area of New Jersey. I will be speaking from a legislator's point of view, while my two colleagues will provide some of the finest technical expertise available in the country today.

They are Dr. A. Judson Wells, who was with the Du Pont Company for 37 years as a scientist and senior executive and who has, since his retirement, been very active in the analysis of the passive smoking issue; and Dr. Ken Sexton, now director of scientific review for the Health Effects Institute in Cambridge, Massachusetts, and formerly, director of the Indoor Air Quality Program for the State of California.

We have helped establish the National Council for Clean Indoor Air because, despite a growing awareness in the science and health fields, indoor air quality is still unknown to much of the general public and to many of your and my colleagues in public office. The council will be bridging that gap, making scientific and medical research available to public policy officials in an understandable and readily available form.

Having been the principal sponsor of the New Jersey radon remediation law, and author of a bill now pending to certify radon inspectors and equipment, I can say that a body of experts such as this council would have been an invaluable asset as we were struggling with this total new public health issue.

As it should be with all new issues, there is an important debate going on regarding the proper role of the Federal Government. There is agreement, however, regarding issues of basic scientific and medical research. Clearly, the Federal Government has a role to play in determining the incidence of the problem, the health effects and the health standards for radon exposure.

Beyond these basics, there is a need for an intensive examination of many of the activities which make up our public policy. Assessment techniques, remedial technologies, examination of radon behavior, financing of remedial actions, public education, and communication between private and public, and State, local, Federal and international agencies are all unsettled issues of responsibility.

Radon in particular and indoor air quality as a whole, are relatively new environmental health issues. But in addition to thrusting in front of us serious matters of public health, these issues also present us with an interesting opportunity.

Indoor air quality issues have little of the baggage attached to them which has now been built around other health and environmental issues. There is the opportunity to start at the beginning and develop a sound marriage of science and public policy and to utilize, in their proper roles, Federal and State action, private industry, and individual initiative.

If we are to achieve this successful blend, indoor air quality must not be allowed to float between agencies. Lead responsibility must clearly be assigned and a long-term commitment to serious budgetary resources must be made.

Management officials at EPA and other Federal agencies, and the policy officials at OMB must understand the congressional commitment to this important public health issue.

In the GAO report on radon to the Pennsylvania congressional delegation, the GAO restated its previous recommendation that EPA be designated the lead agency on indoor air quality issues. The report now adds that formal designation be made for radon as well. Among other things, EPA should be responsible for assuring that (1) radon research efforts do not overlap, (2) radon research is being conducted on the most significant issues, and (3) the Federal Government is operating under and recommending consistent radon policies and guidance. We feel that such a policy statement by this committee would be of great benefit to EPA in creating an effective and efficient radon program.

We would also concur with the GAO finding that formal designation should not interfere with the radon interagency groups which have recently formed. Further, the specific designation of responsibility will make your oversight duties more manageable, accountability more clear, and the desired results more likely.

It is interesting to note that EPA's Science Advisory Board Subcommittee has made similar recommendations to EPA regarding the indoor air quality program as a whole.

I will not go into any more program specifics as I do not want to repeat any of Ken Sexton's overview of indoor air and his discussion of the need for a systematic approach to these many issues, radon included.

As I mentioned in the opening, New Jersey has recently enacted a radon remediation law and is considering further legislation. I won't go into specifics at this time, but would be pleased to answer any questions you may have. I hope we will have the opportunity to discuss some of the things we learned, what we would do differently, what the public reaction has been to this problem, and what the Federal Government provided and what they didn't.

At this time, I would like to give Dr. Wells the opportunity to share with you some of his own research regarding the severity of the passive smoking risk.

Mr. **SCHELUER**. Very good. Dr. Wells.

Mr. **WELLS**. Thank you very much for this opportunity. The area I'm going to talk about today is adult nonsmoker mortality from passive smoking. It's an area I've been studying for—can you hear me all right?

Mr. **SCHEUER**. You mean passive smoking?

Mr. **WELLS**. Passive smoking, right. It's an area I've been studying for the last 5 years, since I retired from Du Pont. And the material I'm presenting today is taken from a paper that I presented at the annual meeting of the air pollution control association, in Minneapolis, last June, and I think you have a copy of the paper there appended to the remarks.

Through the end of 1985, there were 18 epidemiological studies that I knew of that bore on adult nonsmoker mortality from passive smoking. And without going into a lot of detail, by boiling down the data in those studies and combining the data in the various studies, I got some information on four important disease categories for both males and females, and these specifically are lung cancer, cancers other than lung, heart disease, and there was a little bit of data on emphysema.

And if you can find table 1 in those remarks, but I'll summarize it for you anyway, for female lung cancer there were eight studies that I found usable, with an increase in excess risk of 44 percent for those that were exposed to passive smoking. For other cancer for females, there were four studies, with an increase in risk of 56 percent. For heart disease for females, three studies, and an increase in risk of 27 percent. And one study on emphysema with an increase in risk of 40 percent.

For males the data are much scarcer, but there were three studies on lung cancer, with an increase in risk of 150 percent. For other cancer, two studies, and they essentially cancel each other out so there was no effect that you could find in the data so far. And for heart disease, two studies, with an increase in risk of 30 percent.

Now, if you can find table 2 in that bunch of stuff there. By looking at the population exposed and the underlying nonsmoker death rates, it's possible to project these excess risks into an estimate of deaths.

Now I want to emphasize that these are very preliminary numbers. We are talking here about a small number of studies. But nonetheless, if the epidemiology continues along the lines it has taken so far, these numbers do represent more or less the probable numbers you're probably going to be looking at 5 years from now.

Specifically, I come up with about 1,800 deaths a year from lung cancer, and this is right in the middle of the estimate of Repace, and Lowrey, that you may be familiar with, of 500 to 5,000 deaths per year from lung cancer from passive smoking. But when you get into other cancer, you are looking—where you have a much higher underlying nonsmoker death rate, you are looking at something like 20,000 deaths a year. And for heart disease, 25,000 deaths a year.

So I think the way the wind is blowing it looks as though lung cancer is probably just the tip of the iceberg. Now obviously, we need more work to confirm these studies. As I said, they're very preliminary. But we must not be ignoring cancers other than lung and heart disease in our studies of passive smoking.

Another thing that's growing out of this is that the cancer sites seem to be different for passive smoking and direct smoking. If you can find table 3 in there, you'll find that, for example, the entry sites, of course, are different for the two types of smoking. And sure enough, oral cancer shows up for direct smoking, whereas it is absent in passive smoking, and instead you get nasal sinus cancer in passive smoking. There are a large number of what I would call digestive-related cancers in direct smoking that are absent in passive smoking; and, on the other hand, there are cancers like breast, brain and endocrine gland cancers that show up in passive smoking that appear to be absent in direct smoking.

I think, and this is a personal opinion, that the reason for this lies largely in the difference in particle size between the mainstream smoke and the sidestream smoke. Mainstream smoke is a moist, very concentrated aerosol that appears to agglomerate into large particles that deposit mainly in the mouth or in the major airways of the lung. And from these positions it is cleared into the mouth and swallowed, and that results I believe in the cancers that you see on the direct-smoking cancer site list.

In passive smoking the particles are very small and very dilute, and they tend to be deposited either in the nose or they go all the way into the very deep recesses of the lung, the air sacs, or alveoli, as they're called. Clearance from deep in the lung is very slow. It may result in partial solution of these particles and possible clearance directly into the blood or the lymph systems; and, hence, the absence of the digestive cancers and possibly the presence of the cancers that I have enumerated.

To sum up my view of the situation, basically environmental tobacco smoke is a carcinogenic air pollutant. It has been known for years that cigarette smoke is carcinogenic and that it causes heart attacks. Millions of people are exposed to sidestream smoke, and such a material, as Bernard Goldstein has said, is guilty until it is proven innocent, the way we normally go at the regulation of carcinogenic pollutants.

Now the individual victims of passive smoking you are not going to find lying around as corpses on the street corners. It is much like the aftermath of the Chernobyl disaster; they are not going to be identifiable as such. Their numbers are going to be buried in the approximately 1.5 million total heart and cancer deaths in the United States each year. And yet, you know, if we are talking 40,000 deaths a year, that is a large number. Even if we take the 1,800 or so that I have projected for lung cancer, that is a large number, especially when you compare it to the, say, 20 or 100 deaths a year that would attract regulatory attention from any other kind of a carcinogenic air pollutant that we had.

So these results, although, as I've said, are very preliminary, they indicate a strong possibility for a substantial risk from passive smoking, and I believe they call for appropriate Government action.

[The prepared statement of Dr. Wells follows:]

Testimony on
Passive Smoking Mortality
A Review and Preliminary Risk Assessment

by A. Judson Wells, Ph.D.
Vice Chairman

National Council for Clean Indoor Air

before the

Natural Resources, Agriculture Research and Environment
Subcommittee
of the
Committee on Science and Technology
U. S. House of Representatives

September 17, 1986

My name is A. Judson Wells. I am testifying today on behalf of the National Council for Clean Indoor Air. The area I will cover is adult, nonsmoker mortality from passive smoking including a preliminary estimate of the probable number of deaths per year in the U.S. from this cause.

I have a Ph.D. in physical chemistry from Harvard University. My principal career has been in research in the chemical industry, having retired in 1980. For the past five years, on a part time basis, I have been studying the scientific literature on passive smoking with particular reference to mortality effects on adults. The material I am presenting today is taken from a paper I presented at the Annual Meeting of the Air Pollution Control Association in Minneapolis last June. A copy of the paper, updated slightly to clarify some of the tables, is appended to these remarks.

As of last January, when the analysis for the Minneapolis paper was prepared, there were eighteen epidemiological studies that I knew of (I have since found one more) that bore on adult, nonsmoker mortality or cancer morbidity from passive smoking. Five of the eighteen studies were eliminated from the analysis because of (1) inadequate controls, (2) data on current exposure only, (3) insufficient data to calculate a statistical variance, or (4) a serious internal inconsistency. Of the remaining thirteen, eleven dealt with the disease categories of broadest interest, namely, lung cancer, cancers other than lung, ischemic heart disease, and emphysema. If the five papers that were rejected had been included, the results of the analysis would have been essentially unchanged. Also, although I have not yet carried out the mathematics, it is believed that inclusion of the missing 1984 paper and papers that have appeared in 1986 or are about to appear on passive smoking mortality would not have a large effect on the results I will be presenting.

To illustrate the methodology of the epidemiological analysis we will take female lung cancer as an example. Eight of the accepted studies had data on female lung cancer. The relative risks for each study were weighted according to the inverse of the statistical variance for that study. Then the case control data and the cohort study data were each combined separately, and then an overall combination was made. This method was developed at the Harvard School of Public Health. By this procedure the relative risks shown in Table I were obtained for each sex and each disease category. The female "other cancer" risk is based on four studies, the heart disease on three, and the emphysema on one. For the males the lung cancer risk is based on three studies, the other cancer on two and the heart disease on two. Obviously, the male data are much scarcer than the female data. For those of you interested in statistics, the p value is the probability that the result is unity and the confidence interval is the range that the true value falls in with 95% probability.

To develop an estimate of annual deaths from these relative risks it is necessary to have information on the total nonsmoking population, the percent exposed to sidestream smoke, and the death rates for nonsmokers for each disease category. Estimates for these parameters from appropriate sources are shown in Table II along with the relative risks from Table I and the resultant calculated annual deaths by sex and disease category. The estimate for lung cancer, 1800 deaths per year, is right in the middle of the range estimated by Repace and Lowrey of 500 to 5000. However, as can be seen, lung cancer may be just the tip of the iceberg. Other cancer and heart disease, with their much higher underlying nonsmoker death rates and relative risks in the same general range as lung cancer, provide the great bulk of the underlying causes of death. It must be emphasized that these results are preliminary, but if the epidemiology continues the trend it has taken since 1981, these numbers represent the likely outcome.

Another factor that appears to be coming forth from the epidemiology is that the cancer sites for cancers other than lung are different for passive smoking and direct smoking. Based on the few studies that we have so far on specific sites from passive smoking we get the patterns shown in Table III. The entry sites appear to be appropriately different, and direct smoking leads to digestive tract cancers, or close relatives, while passive smoking does not. This difference in sites is thought to arise from a difference in effective particle size between mainstream and sidestream smoke. Mainstream smoke is moist and concentrated and appears to agglomerate into large particles that deposit mainly in the mouth or in the main airways of the lung. From these positions it is cleared into the mouth and swallowed, apparently resulting in the cancers shown. In passive smoking, however, the particles are small and dilute and tend to stay that way. They are breathed through the nose and tend to be deposited either in the nose or deep in the alveolar region of the lung. Clearance from deep in the lung is very slow which may result in partial solution of the particles and clearance into the blood or lymph systems, hence the absence of digestive system cancers and the presence of cancers that may be more blood and lymph related.

Basically, environmental tobacco smoke is a carcinogenic air pollutant. It has been known for years that cigarette smoke is carcinogenic and that it causes heart attacks. Millions of people are exposed (I have noted 32 million (over age 45) in Table II). Such a material, as Bernard Goldstein has said, is guilty until proven innocent. Individual victims of passive smoking, like victims of the aftermath of Chernobyl, will not be identifiable as such. Their numbers will be buried in the approximately 1.5 million total heart and cancer deaths in the U.S. each year. Yet 47,000 preventable deaths is a large number. So is 1800, compared to the 20 to 100 deaths per year that would attract regulatory attention to any other carcinogenic air pollutant. The results, although preliminary, indicate a strong possibility for substantial risk and call for an appropriate government response.

TABLES

for testimony of Dr. A. Judson Wells

September 17, 1986

TABLE I

COMBINED RISK RATIOS FOR PASSIVE SMOKING

Disease	Exposed Cases	R/R*	2-tail p	95% conf. int.
Female				
Lung cancer	463	1.44	<0.001	1.2 - 1.7
Other cancer	2091	1.56	<0.001	1.3 - 1.9
Ischemic heart dis.	276	1.27	0.04	1.0 - 1.6
Emphysema & chr. br.	102	1.4	0.18	0.9 - 2.1
Male				
Lung cancer	13	2.5	0.009	1.3 - 4.7
Other cancer	7	1.0	-	-
Ischemic heart dis.	14	1.3	0.46	0.7 - 2.6

*Risk ratio or relative risk

TABLE II

PRELIMINARY RISK ASSESSMENT FOR PASSIVE SMOKING

PROBABLE NUMBER OF U.S. DEATHS PER YEAR

	Females	Males	Both
Non-smoking Population, 45+	27.6 MM	20.3 MM	47.9
% Exposed	76%	55%	67%
Passive smoking Population, 45+	21.0 MM	11.2 MM	32.2

	Females			Males			Both
Disease	R/R[a]	Non-smoker Death Rate[b]	Deaths from Pass. Smkg.	R/R[a]	Non-smoker Death Rate[b]	Deaths from Pass. Smkg.	Total Deaths
Lung Cancer	1.44	9	600	2.5	13	1,200	1,800
Other Cancer	1.56	245	20,200	1.0	188	0	20,200
Ischemic Heart	1.27	210	9,900	1.3	531	15,300	25,200
Emphysema	1.4	2	100	1.0	6	0	100
Total - four Diseases			30,800			16,500	47,300

[a] Relative risk.

[b] Per 100,000 person years.

TABLE III

CANCER SITE PATTERNS IN DIRECT AND PASSIVE SMOKING

Direct Smoking	Passive Smoking
Buccal Cavity	-
-	Nasal Sinus
Pharynx	-
Larynx	-
Lung - largely bronchial	Lung - largely peripheral
Esophagus	-
Stomach	-
Urinary Bladder	-
Kidney	-
Pancreas	-
-	Breast
-	Brain
-	Endocrine Glands
Cervix	Cervix

RELATIVE RISKS: LUNG CANCER FROM PASSIVE SMOKING

			Highest Exposure		All Exposures		Mantel Trend
Females	Locale	Cases	R/R	2-Tail p	R/R	95% C.L.	1-Tail p
Cohort Studies:							
Hirayama	Japan	163	1.9	0.002	1.6	1.1 - 2.2	0.002
Garfinkel	U. S.	88	1.1	-	1.2	0.8 - 1.6	-
Gillis, et al.	Scotland	6	-	-	1.1	0.2 - 5.6	-
Combined Cohort		257			1.34	1.1 - 1.7	
Case Control:							
Trichopoulos, et al.	Greece	53	2.6	0.19	2.1	1.2 - 3.6	0.005
Correa, et al.	Louisiana	14	3.5	0.017	2.1	0.8 - 5.2	-
Koo, et al.	Hong Kong	64	-	-	1.3	0.7 - 2.3	-
Sandler, et al.	N. Carolina	2	-	-	inf.	-	-
Garfinkel, et al.	U. S.	73	2.0	0.05	1.3	0.8 - 1.9	0.025
Combined C/C		206			1.54	1.2 - 2.0	
Combined Cohort & C/C		463			1.44	1.2 - 1.7	
Males							
Cohort Studies:							
Hirayama	Japan	7	2.3	0.16	2.2	1.1 - 4.8	0.023
Gillis, et al.	Scotland	4	-	-	3.3	0.7-16.4	-
Combined Cohort		11			2.5	1.2 - 5.2	
Case Control:							
Correa, et al.	Louisiana	2	-	-	2.0	0.4-10.0	-
Combined Cohort & C/C		13			2.5	1.3 - 4.7	

RELATIVE RISKS: CANCER OTHER THAN LUNG FROM PASSIVE SMOKING

			Highest Exposure		All Exposures		Mantel Trend
	Locale	Cases	R/R	2-Tail p	R/R	95% C.L.	1-Tail p
Females							
Cohort Studies:							
Hirayama	Japan	1879	1.11	0.055	1.08	1.0 - 1.2	0.05
Gillis, et al.	Scotland	33	-	-	1.2	0.6 - 2.5	-
Combined Cohort		1912			1.1	1.0 - 1.2	
Case Control:							
Miller	Penna.	66	-	-	1.9	1.1 - 3.4	-
Sandler, et al.	N. Carolina	113	-	-	2.0	1.3 - 3.0	-
Combined C/C		179			2.0	1.4 - 2.7	
Combined Cohort & C/C		2091			1.56	1.3 - 1.9	
Males							
Gillis, et al.	Scotland	2	-	-	0.6	0.1 - 2.7	-
Sandler, et al.	N. Carolina	5	-	-	1.5	0.4 - 5.7	-
Combined Cohort & C/C		7			0.9	-	

RELATIVE RISKS FROM PASSIVE SMOKING

ISCHEMIC HEART DISEASE, EMPHYSEMA AND CHRONIC BRONCHITIS

			Highest Exposure		All Exposures		Mantel Trend
	Locale	Cases	R/R	2-Tail p	R/R	95% C.L.	1-Tail p
Heart Dis. - Female							
Cohort Studies:							
Hirayama	Japan	240	1.3	0.038	1.16	0.9 - 1.4	0.02
Gillis, et al.	Scotland	19	-	-	3.6	0.9-13.8	-
Garland, et al.	California	17	-	-	3.5	0.9-13.6	-
Combined		276			1.27	1.0 - 1.6	
Heart Dis. - Male							
Cohort Studies:							
Gillis, et al.	Scotland	14	-	-	1.3	0.7 - 2.6	-
Svendsen, et al.	U.S. (MRFIT)	5	-	-	2.1	0.7 - 6.2	-
Emphysema and Chr. Bronchitis - Female							
Cohort Study:							
Hirayama	Japan	102	1.6	0.08	1.4	0.9 - 2.1	0.05

PASSIVE SMOKING

Specific Cancer Sites Other than Lung

Cancer Site	Investigator(s)	Locale	Cases	R/R
Nasal Sinus	Hirayama	Japan	23	2.1*
Brain	Hirayama	Japan	31	4.5*
Breast:	Hirayama	Japan	-	>1.0
	Sandler, et al.	N. Carolina	32	2.0
Endocrine	Sandler, et al.	N. Carolina	13	4.4*
Cervix:	Buckley, et al.	England	27	4.6*
	Brown, et al.	Canada	30	3.7*
	Sandler, et al.	N. Carolina	56	2.1*
Stomach	Hirayama	Japan	635	1.0

* Some statistical significance claimed by the authors.

DEPOSITION PATTERNS IN DIRECT AND PASSIVE SMOKING

	Direct	Passive
Entry Site	Mouth	Nose
Particulate inhaled, mg. per day	240[1]	0.5 to 3.5[2]
Effective particle size inhaled, μm	5	0.4
Per cent retained in mouth	37	0
Per cent retained in nose	0	8
Per cent retained in bronchus	20	0
% retained in near alveolar region	21	0
Per cent retained in or near alveoli	4	11
Per cent retained, total	82	19
Per cent exhaled	18	81
Particle size exhaled, μm.	0.7	0.4
Particulate retained, mg. per day	197	0.1 to 0.7
Particulate retained in or near alveoli, mg. per day	9.6	0.06 - 0.4

[1] Based on 15 mg. per cigarette and 20 cigarettes per day.

[2] Based on 100-700 μg/m^3 and 10 liters/min. inhaled for 8 hours.

EFFECT OF MISCLASSIFYING SMOKERS AS NONSMOKERS ON PASSIVE SMOKING RELATIVE RISK

Methodology is that of P. Lee, Preventive Medicine 1984; 13:732-3.
Basic assumptions are: 1) 5% Of male and female smokers profess to be nonsmokers.
2) True relative risk of passive smoking is 1.0.

Lee's Lung Cancer Case

	Husbands		
Wives	Smokers	Nonsmokers	Total
Smokers	30%	20%	50%
Nonsmokers	20%	30%	50%
Total	50%	50%	100%

Observed R/R for smokers: 10.7
"True" R/R for smokers: 20
Spurious R/R for passive smokers: 1.42

Realistic Japanese Female Lung Cancer Case

	Husbands		
Wives	Smokers	Never Smkrs	Total
Smokers	15%	1%	16%
Never Smokers	64%	20%	84%
Total	79%	21%	100%

Observed R/R for female smokers: 2.03
"True" R/R for female smokers: 2.05
Spurious R/R for passive smokers: 1.0
Observed R/R for passive smokers: 1.57

Realistic U.S. Female Lung Cancer Case

	Husbands		
Wives	Smkrs	Never Smkrs	Total
Smokers	35%	10%	45%
Never Smokers	30%	25%	55%
Total	65%	35%	100%

Observed R/R for female smokers: 3.58
"True" R/R for female smokers: 4.0
Spurious R/R for passive smokers: 1.1
Observed R/R for passive smokers: 1.44

Realistic U.S. Female Other Cancer Case

	Husbands		
Wives	Smkrs	Never Smkrs	Total
Smokers	35%	10%	45%
Never Smokers	30%	25%	55%
Total	65%	35%	100%

Observed R/R for female smokers: 1.12
"True" R/R for female smokers: 1.126
Spurious R/R for passive smokers: 1.0
Observed R/R for passive smokers: 1.56

APPENDIX

PASSIVE SMOKING MORTALITY

A REVIEW AND PRELIMINARY RISK ASSESSMENT

A. Judson Wells, Ph.D.

Presented at the 79th Annual Meeting
Air Pollution Control Association
Minneapolis, Minnesota
June 24, 1986

Includes minor updating
through September 16,1986

INTRODUCTION

In 1981 when Hirayama[1] and Trichopoulas, et al.[2], first published their studies associating passive smoking with lung cancer, and in the years immediately following, there was considerable controversy and skepticism that such an effect could indeed be real. However, as more papers appeared and many of the earlier issues were resolved, there now appears to be a growing concensus among epidemiologists that passive smoking does indeed cause lung cancer, or at least that there is a strong association. Lawrence Garfinkel, who was one of the most quoted early skeptics, and his coworkers have recently published a paper[3] in which they find a statistically significant doubling of lung cancer risks for women married to smokers of 40 or more cigarettes per day compared to women married to nonsmokers. A dose response relationship was confirmed. Also there is now little disagreement that the number of U.S. deaths from lung cancer associated with passive smoking probably lies within the 500 to 5000 range suggested by Repace and Lowrey's risk assessment[4].

In 1983 papers started to appear associating passive smoking with deaths from other cancers, chronic bronchitis, emphysema and heart disease. These papers, like the earlier lung cancer papers, have attracted debate, but more recent papers support rather than refute the earlier ones, indicating that a consensus eventually may be reached in this broader area as well.

It is the purpose of this paper to present a summary of the epidemiological literature on passive smoking for four major disease categories, namely, lung cancer, other cancer, emphysema, and heart disease, to discuss differences in disease patterns observed between direct and passive smoking and the probable reasons therefor, and to assess at least in a preliminary way the significance of these findings on expected deaths from passive smoking if the underlying epidemiological results turn out to be correct. Because of the many specialties involved in such a calculation, namely, epidemiology, statistics, population estimates of passive smokers, aerosol deposition theory, lung structure, chemistry and carcinogenesis, it is not possible in a paper of this scope to give a detailed and sophisticated explanation of each step. Nonetheless, the estimates made are believed to be the most probable numbers considering the data now available although it must be realized that the confidence limits are still broad because of the uncertainties that exist at every step. The objective here is to determine the probable future extent of the public health risk from passive smoking if the trend in the epidemiological results continues along the lines it has taken since 1981.

METHODS

The epidemiological literature was searched primarily through the publications of the U.S. Government's Office of Smoking and Health[5,6] which also provided a printout of all of its documents on passive smoking from 1970 through 1982. In addition many references were received directly from workers in the field. The scope of the inquiry included all papers that contained original data on adult, nonsmoker mortality or cancer morbidity from passive smoking. All of these papers are listed in the subsequent tables or text. From these papers those pertaining to lung cancer, other cancer, emphysema and heart disease were selected for analysis and calculation of death rates.

Because some of the papers have rather glaring weaknesses, four criteria were used to admit data to the death rate calculations:

1. Retrospective studies should have controls.
2. Observations should be based on exposure beyond five years.
3. A study should not have serious internal inconsistencies.
4. Sufficient data should be presented to allow the calculation of a variance.

For the eleven studies that met these criteria a relative risk for each disease and sex was estimated (to the extent data were available) by averaging over all exposures including exposure to ex-smokers, light, medium and heavy smokers. Combined relative risks for each disease category were calculated by a meta-analysis technique which weights the individual relative risks by the inverse of the variances[7]. No other weighting of the accepted studies was attempted. Some of the cancer papers reported morbidity relative risks rather than mortality relative risks. However, an estimate based on published data[8] indicates that five year survival rates for both exposed and unexposed cases are similar. Therefore, the incidence ratios were used as good approximations to the mortality ratios. The method used to calculate possible deaths from the combined relative risks in the preliminary risk assessment is described under that heading.

RESULTS

Epidemiology

There are eighteen studies now available that bear on adult mortality or cancer morbidity from passive smoking.

Lung Cancer - Female. Table Ia lists the papers on female lung cancer. Shown is the lead author on the paper, the locale of the study, the number of cases, the relative risk for the highest exposure (in most cases 20 or more cigarettes per day smoked by a spouse) and the two-tail p-value for that exposure, a relative risk for all exposures combined, including exposure to ex-smokers if those data are available, and a two-tail p-value for that exposure. A one-tail p-value for a Mantel extension test for trend is shown if available.

TABLE Ia

RELATIVE RISKS: LUNG CANCER FROM PASSIVE SMOKING

	Locale	Exposed Cases	Highest Exposure R/R	Highest Exposure 2-Tail p	All Exposures R/R	All Exposures 95% C.L.	Mantel Trend 1-Tail p
Females							
Cohort Studies:							
Hirayama[20]	Japan	163	1.9	0.002	1.6	1.1 - 2.2	0.002
Garfinkel[11]	U. S.	88	1.1	-	1.2	0.8 - 1.6	-
Gillis, et al.[13]	Scotland	6	-	-	1.1	0.2 - 5.6	-
Combined Cohort		257			1.34	1.1 - 1.7	
Case Control:							
Trichopoulos, et al.[10]	Greece	53	2.6	0.19	2.1	1.2 - 3.6	0.005
Correa, et al.[12]	Louisiana	14	3.5	0.017	2.1	0.8 - 5.2	-
Koo, et al.[14]	Hong Kong	64	-	-	1.3	0.7 - 2.3	-
Sandler, et al.[15]	N. Carolina	2	-	-	inf.	-	-
Garfinkel, et al.[3]	U. S.	73	2.0	0.05	1.3	0.8 - 1.9	0.025
Combined C/C		206			1.54	1.2 - 2.0	
Combined Cohort & C/C		463			1.44	1.2 - 1.7	
Males							
Cohort Studies:							
Hirayama[20]	Japan	7	2.3	0.16	2.2	1.1 - 4.8	0.023
Gillis, et al.[13]	Scotland	4	-	-	3.3	0.7-16.4	-
Combined Cohort		11			2.5	1.2 - 5.2	
Case Control:							
Correa, et al.[12]	Louisiana	2	-	-	2.0	0.4-10.0	-
Combined Cohort & C/C		13			2.5	1.3 - 4.7	

TABLE Ib

RELATIVE RISKS: LUNG CANCER FROM PASSIVE SMOKING

			Highest Exposure		All Exposures		Mantel Trend
	Locale	Exposed Cases	R/R	2-Tail p	R/R	95% C.L.	1-Tail p
Rejected Studies							
Case Control:							
Knoth, et al.[16] (no controls)	W. Germany	24	-	-	2.5	1.0 - 6.4	-
Chan, et al.[17] (current exp. only)	Hong Kong	34	-	-	0.8	0.4 - 1.3	-
Kabat and Wynder[18]							
Females	U.S.	33	-	-	0.5	0.2 - 1.2	-
Males (current exp. only)	U.S.	20	-	-	3.7	1.1-12.7	-
Wu, et al.[19] (insufficient data)	California	?	-	-	1.2	?	-

Data from Dr. Kabat re Kabat and Wynder[18]:

	Females		Males	
Exposure	Cases	Controls	Cases	Controls
Never	20	13	5	12
Work only	17	23	14	10
Home only	7	9	2	2
Work & home	9	8	4	1
Total Exposed	33	40	20	13

There are eight studies that pass the aforementioned criteria. Trichopoulos, et al.[10], have a numerical error in the calculation of relative risks. The values for 1-20 and 21+ cigarettes per day should be 1.95 and 2.55 instead of 2.4 and 3.4, respectively. The value in Table Ia of 2.1 is a weighted average of exposure to ex-smokers as well as to the two categories of smokers. Koo, et al.[14], report on exposures both at home and at work. The work exposures for nonsmokers average only 2.0 years so these data were rejected for being less than five years. Data for exposures at home and at home plus work are combined here to give the relative risk of 1.3 shown in Table Ia. The Sandler, et al.[15], paper was directed largely at total cancer, but they did pick up two lung cancer cases among nonsmokers. These were determined to be female per private communication from Dr. Sandler.

Four of the eight lung cancer studies (Trichopoulos, et al.[10], Correa, et al.[12], Koo, et al.[14], and Sandler, et al.[15]) are disease incidence studies; Hirayama[9], Garfinkel[11] and Gillis, et al.[13], are mortality studies. Garfinkel, et al.[3], is mixed. Since there is no reason to believe that the very low survival rate from lung cancer is significantly different for nonexposed and exposed cases, the incidence relative risks were used as mortality relative risks.

There are four studies that do not pass the criteria, namely, Knoth, et al.[16], which is a case study with no controls, Chan, et al.[17], and Kabat and Wynder[18] which are for current exposure only, and Wu, et al.[19], which does not report the number of cases and hence does not allow the calculation of a variance. The relative risks and significance data for Knoth, Chan and Kabat and Wynder were calculated from data in the papers and from data in a private communication from Dr. Kabat.

Lung Cancer - Male. As shown in Table Ia there are three acceptable passive smoking studies of nonsmoking males with lung cancer. The total of exposed cases is small but the data are consistent. Combined relative risk is 2.5 and p=0.009.

Inclusion of the three rejected female studies for which variances can be calculated and the one rejected male study would raise the combined male relative risk for lung cancer to about 2.7 and would lower the female relative risk to about 1.42. These changes would change the overall death date for lung cancer from passive smoking only slightly and in an upward direction.

Other Cancer - Female. There are now four studies relating passive smoking to cancer other than lung or to total cancer in females (see Table IIa). This relatively new area is more important in terms of potential number of deaths from passive smoking than is lung cancer because the relative risks are in the same range while the underlying death rates for non-smokers are many times higher. The relative risk shown for Hirayama in

TABLE IIa

RELATIVE RISKS: CANCER OTHER THAN LUNG FROM PASSIVE SMOKING

			Highest Exposure		All Exposures		Mantel Trend
Females	Locale	Exposed Cases	R/R	2-Tail p	R/R	95% C.L.	1-Tail p
Cohort Studies:							
Hirayama[9]	Japan	1879	1.11	0.055	1.08	1.0 - 1.2	0.05
Gillis, et al.[13]	Scotland	33	-	-	1.2	0.6 - 2.5	-
Combined Cohort		1912			1.1	1.0 - 1.2	
Case Control:							
Miller[21]	Penna.	66	-	-	1.9	1.1 - 3.4	-
Sandler, et al.[15]	N. Carolina	113	-	-	2.0	1.3 - 3.0	-
Combined C/C		179			2.0	1.4 - 2.7	
Combined Cohort & C/C		2091			1.56	1.3 - 1.9	
Males							
Gillis, et al.[13]	Scotland	2	-	-	0.6	0.1 - 2.7	-
Sandler, et al.[15]	N. Carolina	5	-	-	1.5	0.4 - 5.7	-
Combined Cohort & C/C		7			0.9	-	

TABLE IIb

RELATIVE RISKS FROM PASSIVE SMOKING

ISCHEMIC HEART DISEASE, EMPHYSEMA AND CHRONIC BRONCHITIS

	Locale	Exposed Cases	Highest Exposure R/R	Highest Exposure 2-Tail p	All Exposures R/R	All Exposures 95% C.L.	Mantel Trend 1-Tail p
Heart Dis. - Female							
Cohort Studies:							
Hirayama[9]	Japan	240	1.3	0.038	1.16	0.9 - 1.4	0.02
Gillis, et al.[13]	Scotland	19	-	-	3.6	0.9-13.8	-
Garland, et al.[22]	California	17	-	-	3.5	0.9-13.6	-
Combined		276			1.27	1.0 - 1.6	
Heart Dis. - Male							
Cohort Studies:							
Gillis, et al.[13]	Scotland	14	-	-	1.3	0.7 - 2.6	-
Svendsen, et al.[23]	U.S. (MRFIT)	5	-	-	2.1	0.7 - 6.2	-
Emphysema and Chr. Bronchitis - Female							
Cohort Study:							
Hirayama[9]	Japan	102	1.6	0.08	1.4	0.9 - 2.1	0.05

Table IIa of 1.08 is obtained by combining his values for higher and lower exposures. The Miller[21] result is for total cancer. Our interest here is in a relative risk for cancer other than lung, not total cancer. However, according to the calculation described later, less than 3% of Miller's total cancer cases should be lung cancer, and the relative risks for lung and total cancer are similar. Therefore, his total cancer relative risk is a good approximation to the relative risk for cancer other than lung. The paper by Sandler, et al.[15], is also directed at total cancer. Here the number of lung cancer cases is known to be two. Therefore, again the total cancer relative risk is a good approximation to other cancer relative risk.

Other Cancer - Male. The data for cancer other than lung for males (See Table IIa) are much scarcer than for females. The results from the two available papers[13,15] are conflicting, neither result is statistically significant nor is the combined relative risk. Therefore, a value of 1.0, indicating no association, is assumed for the death rate calculations until more data become available.

Heart Disease - Female. There are now three papers associating passive smoking with heart disease among females (see Table IIb). Hirayama's paper[9] contains data for ischemic heart disease in women by smoking habit of the husbands for two levels of exposure. The relative risk for all exposures of 1.16 is a weighted average of the two exposure levels. Gillis, et al.[13], report data for myocardial infarction (MI-410) and for other ischemic heart disease (IHD-411-414). These have been combined to yield the ischemic heart disease relative risk of 3.6 shown in Table IIb. Garland, et al.[22], report data for nonsmoking women who were married either to nonsmokers, ex-smokers, or current smokers. The overall relative risk of 3.5 in Table IIb is calculated from a weighted average of their age adjusted mortality rates for exposure to ex-smokers and smokers.

Heart Disease - Male. There are two papers associating passive smoking with heart disease in males. One, shown in Table IIb is Gillis, et al.[13] Their relative risk of 1.3, although not statistically significant, is reinforced by Svendsen, et al.[23], who found a relative risk of 2.12 ($p=0.19$) for the MRFIT cohort of relatively high risk individuals. Since the two papers concur and there are no negative or neutral results, the Gillis, et al. result, 1.3, is used as the passive smoking relative risk for male ischemic heart disease in the general population until better data become available.

Emphysema and Chronic Bronchitis. Hirayama[9] has the only data on these diseases, and they are for females only. As shown in Table IIb, a relative risk of 1.4 is obtained by combining his results for high and low exposures.

Specific Cancer Sites Other than Lung. Table III shows results on passive smoking risks for specific cancer sites other than lung. It is interesting that the sites other than lung that are normally associated with direct smoking are absent, with the possible exception of cervix. The data on breast cancer are very preliminary. Hirayama [20] reported that risk elevation for all cancer sites becomes non-significant when lung, nasal sinus, brain and breast are excluded. The Sandler, et al. [15], relative risk is of borderline statistical significance. It is interesting that Hirayama [20] found stomach cancer, with 635 cases, to be specifically not associated with passive smoking.

Table III. Epidemiological studies on passive smoking Specific cancer sites other than lung.

Cancer Site	Investigators	Locale	Cases	R/R
Nasal Sinus	Hirayama 20	Japan	23	2.1a
Brain	Hirayama 20	Japan	31	4.5a
Breast:				
	Hirayama 20	Japan	-	>1.0
	Sandler, et al. 15	N. Carolina	32	2.0
Endocrine	Sandler, et al. 15	N. Carolina	13	4.4a
Cervix:				
	Buckley, et al. 24	England	27	4.6a
	Brown, et al. 25	Canada	30	3.7a
	Sandler, et al. 15	N. Carolina	56	2.1a
Stomach	Hirayama 20	Japan	635	1.0

a Some statistical significance claimed by authors.

All Causes of Death. There are five studies that contain data on passive smoking and all causes of death. The relative risks are diluted by a large number of deaths that are not related to passive smoking at all so the results are scattered and lower. The Miller study [26] in 1978 is the pioneer study on mortality from passive smoking. He observed a lowering of life expectancy from 78.8 years to 74.7 years for 601 non-smoking wives whose husbands smoked compared with those whose husbands did not smoke. Gillis, et al. [13], observed a relative risk of 1.5 (81 cases, p=0.17) for females and 1.0 (28 cases) for males. Garland, et al. [22], observed a relative risk of 1.05 (57 cases) for females. Svendsen, et al. [23], observed a relative risk of 2.0 (11 cases, p=0.07) for males in the MRFIT cohort. The only negative relative risk comes from Vandenbroucke, et al. [27], in Holland (0.8 for females; 207 cases, p=0.12). However, this study is flawed in that non-exposed non-smokers had a death rate 15% higher than direct smokers so it would not pass criterion number three that a study should not have a serious inconsistency.

Having reviewed the mortality literature on passive smoking, what does it all mean? First, there is quite a bit of it. Second, taken as a whole there is growing evidence of an association between passive smoking and fatal disease. The lung cancer data are particularly persuasive, and there are preliminary data that indicate an association with total cancer, ischemic heart disease and emphysema and chronic bronchitis. Third, the cancer patterns in direct smoking and passive smoking appear to be different.

Cancer Sites

Table IV shows a comparison of cancer sites for direct smoking (from reference 28), and passive smoking (from Table III). It is evident that the entry sites are different, there is a qualitative difference in the location of the lung cancer sites [29,30] , and the sites elsewhere in the body (except for cervix) are different. Possible reasons for these differences are discussed later. One conclusion that can be drawn, however, is that passive smokers are not a mixture of true non-smokers and a small percentage of misclassified direct smokers. Such a mixture would exhibit the same pattern of excess disease rates as direct smokers except that the increases in relative risks would be smaller.

Table IV. Cancer site patterns in direct and passive smoking

Direct Smoking	Passive Smoking
Buccal Cavity	-
-	Nasal Sinus
Pharynx	-
Larynx	-
Lung - largely bronchial	Lung - largely peripheral
Esophagus	-
Stomach	-
Urinary Bladder	-
Kidney	-
Pancreas	-
-	Breast
-	Brain
-	Endocrine glands
Cervix	Cervix

Particle Size Effects

Probably the most important difference between mainstream and sidestream smoke is the difference in effective particle size and its effect on where the smoke particles are deposited. A careful study of the smoke and aerosol deposition literature discloses a number of differences which are summarized in Table V.

Direct smokers retain about 82% [31] of the inhaled particulate of which about 37% is retained in the buccal cavity and 45% in the lower respiratory tract. Although particle size measurements made as mainstream smoke is generated indicate a mass median aerodynamic diameter of about 0.5μm, the particles, in the moist, high concentration conditions of direct smoking, apparently agglomerate to a much larger effective particle size. To exhibit deposition behavior such as that cited above the effective particle size would have to be in the 5μm range[33]. This increase could be brought about either by direct agglomeration[34], by electrical charges generated on the particles[35], by a dense layering effect[36], or by some combination of these. Particles of this effective size would deposit heavily in the larger bronchial airways[37], particularly at the bifurcations, and in the larger airways of the alveolar region. The exhaled smoke has a particle size of about 0.7μm[31]. Only about one-fifth of this size would be deposited, being 25% of the 18% exhaled or about 4% of the total inhaled, and this would come down deep in the alveolar region, in or near the alveoli themselves[33,37].

Table V. Deposition patterns in direct and passive smoking.

	Direct	Passive
Entry Site	Mouth	Nose
Particulate inhaled per day, mg.	240[a]	0.5 to 3.5[b]
Effective particle size inhaled, μm.	5	0.4
Percent retained in mouth	37	0
Percent retained in nose	0	8
Percent retained in bronchus	20	0
Percent retained in near alveolar region	21	0
Percent retained in or near alveoli	4	11
Percent retained, total	82	19
Percent exhaled	18	81
Particle size exhaled, μm	0.7	0.4

[a] Based on 15 mg. per cigarette and 20 cigarettes per day.
[b] Based on 100-700 $\mu g/m^3$[32] and 10 liters/min. inhaled for 8 hrs.

In contrast sidestream smoke is very dilute. It has a mass median aerodynamic diameter of about 0.4μm[38] and does not agglomerate. This is a very difficult size to trap in the respiratory tract because it is too large to deposit by diffusion and too small to deposit by impaction. Some of the larger particles in the sidestream smoke (about 8% of the amount inhaled) would be deposited in the nose[33]. The other 11% that will deposit[38] apparently goes all the way through the bronchial and larger alveolar airways and deposits in or near the alveoli[33,37].

The particulate from direct smoking is either deposited in the mouth directly, or in the bronchial region or near alveolar region where it will be cleared into the mouth. It is then swallowed and is either eliminated or absorbed into the blood stream via the gut. This is believed to result in the cancers shown in Table IV except possibly for cervix. The particulate from passive smoking deposits either in the nose, resulting in nasal sinus cancer, or deep in the alveoli from whence it is very difficult to clear into the mouth. It is speculated that most of this particulate is solubilized or metabolized directly into the blood or lymph system. It then circulates in these systems and results in the cancers observed. In passive smoking the digestive related cancers are absent.

Chemistry and Other Effects

Another difference between mainstream and sidestream smoke is the chemistry. Side stream smoke is formed at a lower temperature than mainstream smoke, and, therefore, a larger fraction of the more complex molecules is preserved. The amounts of tumorigenic agents in sidestream and mainstream smoke have been measured 39. The sidestream/mainstream ratios for such amounts vary from 0.7 for catechol to 39 for 2-naphthylamine. The mean value is 12. Therefore, it would be expected that sidestream smoke particulate, per milligram deposited, would have higher carcinogenic potential than mainstream particulate.

Another difference between direct smoking and passive smoking is the difference in disease susceptibility between the direct and passive smokers. Deaths attributed to direct smoking constitute about 18% of total deaths. Thus a direct smoker dying of a smoking related disease, while perhaps more sensitive than the average of the total population, would not be substantially more sensitive. Passive smokers dying of passive smoking disease, on the other hand, constitute only a very small percentage of total deaths, in the range of 0.025 to 2.5%. This is a much smaller proportion than in the case of direct smoking; only the very most sensitive individuals would be dying from such an effect, and the expected dose to achieve such a response would be less than that for a direct smoker.

One approach to predicting the health effects of passive smoking has been to factor down the health effects of direct smoking by the ratio of inhaled or deposited smoke dose. As can be seen from the data in Table V the dose ratio is quite high, being about 70 to 500 for inhaled dose and about 300 to 2100 for deposited dose. However, taking into account the differences in particle size effects, chemistry and individual susceptibility, it is seen that such a simplistic dose/response approach is unlikely to yield results that are accurate even within an order of magnitude.

Risk Assessment

Just how dangerous might passive smoking be? Table VI provides a summary of the combined relative risks for each sex and disease. Probably the most reliable number in the table is the 1.44 relative risk for female lung cancer, based, as it is, on eight studies including those by some of the most prominent investigators in the field. The least reliable numbers are those for male other cancer, where an arbitrary relative risk of 1.0, denoting no association, was adopted, and for male ischemic heart disease. Clearly more work is needed in these areas. The relative risk for emphysema and chronic bronchitis also has poor statistical significance, but the underlying death rates for non-smokers from these diseases is so low that only a very few deaths would be involved.

Table VI. Combined Risk Ratios from Epidemiological Studies

Disease	Cases	R/R	2-tail p	95% conf. int.
Female:				
Lung cancer	463	1.44	<0.001	1.2 - 1.7
Other cancer	2091	1.56	<0.001	1.3 - 1.9
Ischemic heart dis.	276	1.27	0.04	1.0 - 1.6
Emphysema & chr. br.	102	1.4	0.18	0.9 - 2.1
Male:				
Lung cancer	13	2.5	0.009	1.3 - 4.7
Other cancer	7	1.0	-	-
Ischemic heart dis.	14	1.3	0.46	0.7 - 2.6

To calculate numbers of deaths per year from the relative risks in Table VI it is necessary to know the total non-smoking population, the percent exposed to sidestream smoke and the death rates for non-smokers by disease category. The non-smoker population was estimated from the national health statistics. The percent of non-smokers exposed to spouse's smoke was estimated from the controls in the U.S. studies in Tables I and II. Other exposure was estimated using data developed by Friedman, et al.[40] Non-smoker death rates for each sex and disease were obtained from Hammond [41]. By combining these inputs it was possible to calculate excess death rates due to passive smoking, again by sex and disease. Applying these death rates to the non-smoking population passively exposed, the desired number of deaths per year in the U.S. was obtained. Details of this calculation are in a manuscript that has been submitted for publication elsewhere.

By this procedure deaths from passive smoking for lung cancer for males plus females came to 1800 per year, well centered in the range of 500 to 5000 obtained in a previous study . For other cancer and heart disease the relative risks are in the same range as lung cancer (Table VI), but the underlying non-smoker death rates are much higher. Therefore, the estimated deaths are much higher. The total deaths came to 47,000 per year of which 22,000 were from cancer (including the 1800 lung cancer deaths) and 25,000 were from heart disease. This assumes no excess deaths from male cancer other than lung but does include deaths from male ischemic heart disease at a relative risk of 1.3. This value, as noted earlier, is supported by Svendsen, et al[23], and is very close to the female relative risk of 1.27. Even if these male deaths are excluded, the total passive smoking deaths would still calculate out to 32,000 per year.

CONCLUSIONS

The epidemiological literature on mortality from passive smoking is growing. An association between passive smoking and lung cancer is becoming increasingly evident, and there are beginnings, at least, of evidence that other cancers and heart disease are also involved. If these trends continue, lung cancer will become only the tip of the iceberg with deaths from these latter diseases amounting to ten to twenty times those from lung cancer. Tobacco smoke is known to be carcinogenic and to produce heart disease. Millions of non-smokers, estimated in this study at 32 million, are exposed. What we have, in other words, are the slowly emerging shapes and dimensions of a major public health problem. Furthermore it is an indoor problem. That means that it is a home-oriented and workplace problem because that is where the average passive smoker spends most of his or her time. There may not be much that can be done about the home setting, but officials and managers who are responsible for worksites need to become aware of this increasingly acknowledged threat to the safety of indoor air and the workers who are exposed to it. Also this is not a traditional workplace air pollutant that emanates from some facet of the work itself and on the factory floor. Rather it occurs in offices, smoke break rooms, washrooms and other places that have always been considered safe; and the pollutant arises not from the work itself but from the other workers. The first action required is to protect the non-smokers from the smokers. The next thing to consider is a smoke-free workplace.

ACKNOWLEDGEMENTS

The author wishes to acknowledge major assistance from Graham A. Colditz, M.D., who did the statistical meta-analysis, and very helpful suggestions from William J. Blot, Ph.D., Kenneth P. Cantor, Ph.D., Norman H. Edelman, M.D., Edwin B. Fisher, Ph.D., F. Charles Hiller, M.D., James L. Repace, Ph.D., Jonathan M. Samet, M.D., Dale P. Sandler, Ph.D., and Frank E. Speizer, M.D.

This work was supported in part by the American Lung Association. The opinions expressed are those of the author. No official endorsement by the American Lung Association should be inferred.

REFERENCES

1. T. Hirayama, "Non-smoking wives of heavy smokers have a higher risk of lung cancer: a study from Japan", *Br. Med. J.* *282*:183 (1981).
2. D. Trichopoulos, A. Kalandidi, L. Sparros, B. MacMahon, "Lung cancer and passive smoking", *Int. J. Cancer* *27*:1 (1981).
3. L. Garfinkel, O. Auerbach, L. Joubert, "Involuntary smoking and lung cancer: a case control study", *J. Nat. Cancer Inst.* *75*:463 (1985).
4. J. L. Repace, A. H. Lowrey, "A quantitative estimate of nonsmokers' lung cancer risk from passive smoking", *Environ. Int.* *11*:3 (1985).
5. *Bibliography on smoking and health*, Public Health Service Bibliography Series No. 45, Public Health Service, Rockville, MD, 1979, 1980, 1981, 1982, 1983, 1984.
6. *Smoking and health bulletin*, Public Health Service, Rockville, MD, 1985 issues.
7. K. T. Halvorsen, *Estimating population parameters using information from several independent sources*, Doctoral dissertation. Harvard School of Public Health, Boston, MA, 1984, p. 46.
8. *Cancer Facts and Figures, 1984*, American Cancer Society, New York, 1984, p. 7.
9. T. Hirayama, "Passive smoking and lung cancer", presented at the Fifth World Conference on Smoking and Health, Winnepeg, Canada, July, 1983.
10. D. Trichopoulos, A. Kalandidi, L. Sparros, "Lung cancer and passive smoking: conclusion of Greek study", *Lancet* *2*:677 (1983).
11. L. Garfinkel, "Time trends in lung cancer mortality among nonsmokers and a note on passive smoking", *J. Nat. Cancer Inst.* *66*:1061 (1981).
12. P. Correa, L. W. Pickle, E. Fontham, Y. Lin, W. Haenszel, "Passive smoking and lung cancer", *Lancet* *2*:595 (1983).
13. C. R. Gillis, D. J. Hole, V. M. Hawthorne, P. Boyle, "The effect of environmental tobacco smoke in two urban communities in the west of Scotland", *Eur. J. Resp. Dis.* *65* (supplement No. 133):121 (1984).
14. L. C. Koo, J. H-C. Ho, D. Saw, "Is passive smoking an added risk factor for lung cancer in Chinese women?" *J. Exp. Clin. Cancer Res.* *3*:277 (1984).
15. D. P. Sandler, R. B. Everson, A. J. Wilcox, "Passive smoking in adulthood and cancer risk", *Am. J. Epidemiol.* *121*:37 (1985).
16. A. Knoth, H. Bohn, F. Schmidt, "Passivrauchen als Lungenkrebs-Ursache bei Nichtraucherinnen", *Medizinische Klinik* *78*:66 (1983).

17. W. C. Chan, M. J. Colbourne, S. C. Fung, H. C. Ho, "Bronchial cancer in Hong Kong 1976-1977", Br. J. Cancer 39:182 (1979). See also W. C. Chan, S. C. Fung, "Lung cancer in non-smokers in Hong Kong", in Cancer campaign, Vol. 6, Cancer Epidemiology, E. Grundmann, ed., Gustav Fischer Verlag, Stuttgart, New York, 1982, pp. 199-202, which appears to be based on the same data.
18. G. C. Kabat, E. L. Wynder, "Lung cancer in nonsmokers", Cancer 53:1214 (1984).
19. A. H. Wu, B. E. Henderson, M. C. Pike, M. C. Yu, "Smoking and other risk factors for lung cancer in women", J. Nat. Cancer Inst. 74:747 (1985).
20. T. Hirayama, "Cancer mortality in nonsmoking women with smoking husbands based on a large-scale cohort study in Japan", Preventive Med. 13:680 (1984).
21. G. H. Miller, "Cancer, passive smoking and non-employed and employed wives", West. J. Med. 140:632 (1984).
22. C. Garland, E. Barrett-Connor, L. Suarez, M. H. Criqui, D. L. Wingard, "Effects of passive smoking on ischemic heart disease mortality of nonsmokers", Am. J. Epidemiol. 121:645 (1985).
23. K. H. Svendsen, L. H. Kuller, J. D. Neaton, "Effects of passive smoking in the multiple risk factor intervention trial (MRFIT)", presented at American Heart Association Meeting, Washington, D.C., November, 1985.
24. J. D. Buckley, R. W. C. Harris, R. Doll, M. P. Vessey, P. T. Williams, "Case-control study of the husbands of women with dysplasia or carcinoma of the cervix uteri", Lancet 2:1010 (1981).
25. D. C. Brown, L. Pereira, J. B. Garner, "Cancer of the cervix and the smoking husband", Can. Fam. Physician 28:499 (1982).
26. G. H. Miller, "The Pennsylvania study on passive smoking", J. Breathing, Illinois Lung Assoc. 41(5):5 (1978).
27. J. P. Vandenbroucke, J. H. H. Verheesen, A. De Bruin, B. J. Mauritz, C. Van Der Heide-Wessel, R. M. Van Der Heide, "Active and passive smoking in married couples: results of 25 year followup", Br. Med. J. 288:1801 (1984).
28. The health consequences of smoking, cancer, a report of the surgeon general, 1982, Public Health Service, Rockville, MD, 1982, pp. 145-6.
29. L. H. Garland, R. L. Beier, W. Coulson, J. H. Heald, R. L. Stein, "The apparent sites of origin of carcinomas of the lung", Radiology 78:1 (1962).
30. R. G. Vincent, J. W. Pickren, W. W. Lane, I. Bross, H. Takita, L. Houten, A. C. Gutierrez, T. Rzepka, "The changing histopathology of lung cancer, a review of 1682 cases", Cancer 34:1647 (1977).
31. R. J. Mitchell, "Controlled measurement of smoke-particle retention in the respiratory tract", Am. Rev. Respir. Dis. 85:526 (1962).
32. J. L. Repace, A. H. Lowrey, "Indoor air pollution, tobacco smoke, and public health", Science 208:464 (1980).

33. J. Heyder, "Studies of particle deposition and clearance in humans", in *Problems of inhalatory toxicity studies*, P. Grosdanoff, et al., eds., MMV-Medizin-Verlag, Munich, 1985, pp. 155-180.

34. K. McCusker, C. Hiller, M. Mazumder, R. Bone, "Dynamic growth of cigarette smoke particles ", (Abstract), *Chest* *80*:349 (1981).

35. C. Melandri, G. Tarroni, V. Prodi, T. De Zaiacomo, H. Formignani, C. C. Lombardi, "Deposition of charged particles in the human airways", *J. Aerosol. Sci.* *14*:657 (1983).

36. N. A. Fuchs, *The mechanics of aerosols*, Macmillan, New York, 1964, pp. 46-49.

37. T. R. Gerrity, P. S. Lee, F. J. Hass, A. Marinelli, P. Werner, R. V. Lourenco, "Calculated deposition of inhaled particles in the airway generations of normal subjects", *J. Appl. Physiol: Respirat. Environ. Exercise Physiol.* *47*(4):867 (1979).

38. F. C. Hiller, K. T. McCusker, M. K. Mazumder, J. D. Wilson, R. C. Bone, "Deposition of sidestream cigarette smoke in the human respiratory tract", *Am. Rev. Resp. Dis.* *125*(4):406 (1982).

39. *The health consequences of smoking, cancer, a report of the surgeon general, 1982*, Public Health Service, Rockville, MD, pp. 196-214.

40. G. D. Friedman, D. B. Petitti, R. D. Bawol, "Prevalence and correlates of passive smoking", *Am. J. Public Health* *73*:401 (1983).

41. E. C. Hammond, "Smoking in relation to the death rates of one million men and women", in *Epidemiological approaches to the study of cancer and other chronic diseases*, W. Haenszel, ed., Public Health Service, Bethesda, MD, 1966, pp. 127-204. (National Cancer Institute Monograph 19).

Mr. SCHEULER. Very good. Thank you very much. Dr. Sexton.

Dr. SEXTON. Thank you. I will take you at your word about being informal and just try very briefly to put maybe the issue in perspective a little bit and try to highlight a couple of issues that weren't touched on very greatly here today.

It seems to me that the question that we're faced with is now that we've discovered indoor air pollution, what are we going to do about it? And as we've heard here today, we've heard a lot of talk about indoor air quality as a generic issue. And in fact, it is not a generic issue. There are many different kinds of indoor environments, and most of what we talked about here today it seems to me has been the residential indoor environment. And that has really not been made explicit, but that has been the focus.

In point of fact, there are indoor air quality problems in many different kinds of buildings, and we tend to know less about the indoor air quality problems in office buildings, commercial buildings, schools, for instance, than we do even in the home.

Also, I think that the different kinds of indoor environments that we have have different policy ramifications. It seems to me that Government's responsibility might be quite different in a person's home than it might be in the workplace, for instance, in an office building. In a person's home, people have some direct control over their own indoor air quality, and perhaps personal choices are something that we would want to foster, we would want to provide information. In the workplace, in an office, for instance, it might be that Government has a more direct role. Perhaps specifying minimum ventilation rates or even guidelines or regulations.

I think these policy questions are important, and, perhaps, as important, if not more important, than the technical questions. Because the way we answer some of these policy questions I feel is going to direct the way we do our research.

I would also like to point out what I think is a very important ramification of indoor air quality, and that is, first of all, that we have pollutants, mostly that we have talked about today, that are indoor pollutants. By that I mean they arise from indoor sources. They are a problem only inside buildings. We've talked about several of them today: asbestos, radon, microbials, tobacco smoke. Formaldehyde tends to be an indoor air pollution problem, and many volatile organic compounds, as near as we can tell, seem to be primarily an indoor problem.

We have other kinds of pollutants that have both indoor and outdoor sources. The Clean Air Act—as you know, the Clean Air Act is written specifically for outdoor air, but there at least three pollutants for which there are criteria standards—carbon monoxide, nitrogen dioxide, and particles—that have both indoor and outdoor sources. And the fact that we rely on outdoor monitors to provide us with data on compliance with our air pollution health laws we now know to be a problem. We know that these outdoor monitors do not provide very good exposure data even for these pollutants that have both indoor and outdoor sources. I think that is a very important to make; that we shouldn't focus just on the indoor pollutants, those that have indoor sources, but there are ramifications here for other air pollution programs and which we are undertaking.

I would also say, in summary, that it seems to me that we've, basically, in the indoor air quality field, have done hazard identification. We've identified some potential hazards. On some pollutants we have gone further than others. We have a pretty good idea of significant health risks. That we are really at the point where I think we've heard today where we need to step back, take a close look and design a comprehensive and systematic program to answer some of these important questions.

Thank you.

[The prepared statement of Dr. Sexton follows:]

Testimony by

Ken Sexton, Sc.D.
Director for Scientific Review
Health Effects Institute
Cambridge, Massachusetts 02142

Subcommittee on Natural Resources,
Agriculture Research, and the Environment

Committee on Science and Technology
House of Representatives

September 17, 1986

SAFEGUARDING INDOOR AIR QUALITY

PUBLIC POLICY ISSUES

Providing and maintaining healthful air quality in nonindustrial indoor environments is more than just a complex technical issue. Realization that contaminated indoor air may pose a significant health hazard raises complicated public policy questions about the proper role of government in safeguarding public health inside private and public buildings. Although little effort has been devoted to identifying, let alone resolving, the critical policy questions, it is becoming increasingly apparent that this process must move forward before meaningful progress can be made in ameliorating indoor air quality problems. Here are some of the major questions that must be addressed:

- Does the role of government depend on the degree of "publicness" for a particular building? And if so, what is an appropriate response to air quality problems in private, as opposed to public, buildings?

- Is consideration of the difference between voluntary and nonvoluntary risks important for choices about government intervention?

- What are the trade-offs (e.g., costs versus benefits) between energy conservation measures and healthful indoor air quality?

- Should the emphasis be placed on protecting building occupants from long-term chronic exposures or short-term peak exposures?

- Should everyone, including sensitive individuals (e.g., infants, the elderly, those with asthma and emphysema), be afforded the same degree of protection from indoor air contaminants?

- What are the responsibilities incumbent upon different sectors of society (e.g., individuals, building owners and operators, architects, developers, contractors, manufacturers, government), which have a stake in the issue?

- If it is decided that government intervention is justified, what forms of intervention are appropriate?

- If government actions to ameliorate problems are deemed necessary, at what level of government (i.e., local, state, or Federal) should authority and responsibility be vested?

POTENTIAL GOVERNMENT RESPONSES

Although scientists and health officials have come to realize that contaminated air inside buildings is a major exposure route for certain air pollutants, policymakers must deal with the question of how best to protect public health in the face of incomplete and sometimes contradictory information. Moreover, workable strategies to mitigate indoor air quality problems must balance the need to protect individual privacy against government's responsibility to protect citizens' health and safety. But, to date, there has been no systematic evaluation of governmental options for dealing with unhealthful indoor environments.

Resolution of the important public policy issues will obviously affect decisions about the appropriate forms of intervention. For example, if it is determined that air quality inside private residences is a matter of personal choice (as public policy so states for saccharin and cigarettes), then government efforts to ameliorate health hazards should focus on public information programs and product labeling requirements to ensure that individuals have sufficient information to make informed choices. Likewise, if air quality inside offices and schools is deemed to be a "public good" (implying that air pollution exposure in these indoor environments represents an involuntary risk over which building occupants have little or no control), then government actions might include rules and regulations to protect public health. A number of potential governmental responses to indoor air quality problems come to mind. The government could

- take no action, based on findings of inconsequential public health risks or unfavorable cost/benefit ratios;
- encourage more research to fill critical knowledge gaps that are central to decisions about the necessity of government intervention;
- treat as a personal choice issue (e.g., as with saccharin or cigarettes) and emphasize public information programs and product labeling;
- institute economic incentives or disincentives to encourage maintenance of adequate indoor air quality;
- use moral suasion to promote voluntary industry codes and standards that reduce indoor exposures;
- define legal responsibility and liability for safe and healthful indoor environments;

- establish exposure guidelines to aid individuals, professionals, and regulatory officials in achieving and maintaining safe indoor air quality;

- promulgate rules and regulations, including design standards (e.g., building codes, minimum ventilation requirements), emission standards (e.g., emission limitations for appliances, consumer products, building materials), and indoor air quality standards (e.g., specify maximum allowable concentrations of important contaminants).

REGULATORY ISSUES

Creation of a regulatory framework for protection of indoor environmental quality poses special policy issues that bear directly on choices about appropriate public responses. Promulgation of indoor air quality standards and other regulations must acknowledge that individuals (especially in private residences) are already making decisions about their own air quality. Development of effective and reasonable policy requires an appreciation of the scope for private action, as well as consideration of the likelihood that public intervention will foster improved private choices.

This is not to suggest that rules and regulations have no part to play in safeguarding indoor air quality. This form of intervention is, however, not necessarily optimal or even desirable in certain types of indoor environments. Although indoor air quality is often spoken of in a generic sense, there are, of course, different types of indoor settings--for example, (1) occupational, both industrial and nonindustrial, (2) nonoccupational, including residential, commercial, institutional, and public, and (3) transportation microenvironments, such as automobiles, airplanes, subways, and trains.

The role of government varies according to the "publicness" of a particular space as well as according to the nature of air pollution health risks, either voluntary or nonvoluntary. Understanding the diversity of nonindustrial indoor environments will be an important preliminary to the design and implementation of practical and cost-effective control stategies.

The rationale for government regulation of outdoor air pollution is based in part on a definition of outdoor air as a "public good" and on the realization that those who suffer the effects of such pollution are neither compensated by nor powerful in influencing polluters. The situation is quite different for some indoor environments, especially private residences, for both the costs and benefits of pollution control are internalized within households.

If occupants foul the air in their home, they are forced to breathe it. If they attempt to improve its quality by increasing ventilation or installing air-cleaning devices, they bear the costs and enjoy the benefits. For some contaminants, such as tobacco smoke, odorants, and water vapor, benefits are readily recognizable through improvements in perceptible air quality and reduction of corrosion, soiling, and molds. Benefits will include reduced health risks due to lower exposures for those pollutants harmful to human health, but below perception thresholds.

The closed-loop cost-benefit cycle within residences suggests that individual decisions are important determinants of indoor air quality. However, unlike residential energy consumption, where monthly bills from the local utility company provide periodic feedback to consumers, indoor contaminants may be below irritation or odor thresholds. Thus, although individuals are already making decisions about their own air quality, it is not clear that these are "informed" decisions. Among the factors likely to affect individual choices are perceived costs and benefits, susceptibility to pollutants, awareness of health risks, and perceptions of existing indoor air quality. In private residences, government actions aimed at improving personal decisions about indoor air quality may be preferable to rules and regulations (e.g., simple warning devices, product labeling, or information programs).

Not all buildings are residences, and not all residences are owner-occupied. The rational for direct government intervention aimed at improving indoor air quality (e.g, rules and regulations) is much stronger in public, as opposed to private, buildings. Air quality in large public buildings, for instance, has many characteristics of a public good. A person sensitive to tobacco smoke would not rationally pay the costs of cleaning the air in a large convention hall. The costs would greatly exceed any personal benefits an individual might derive from smoke-free air, and those who did not contribute could not be excluded from enjoying the benefits. In this situation, indoor air has many characteristics of a public good and the rationale for regulation is similar to that for outdoor air pollution.

There is also substantial justification for regulatory intervention in private and public buildings where occupants do not have control over their own environment--for example, modern high-rise office buildings. Typically, building managers are responsible for operation and maintenance of heating, ventilation, and air-conditioning (HVAC) systems. Occupants of the building, including both employers and employees, often have lttle or no direct control of temperature, fresh air input, and ventilation rate. Because HVAC systems are normally operated to minimize energy costs (a parameter easily monitored through utility bills), the health and comfort of tenants (ambiguous parameters that are difficult to quantify) rarely become an issue unless a significant number of complaints are reported.

Because health risks in this situation tend to be nonvoluntary, government has a responsibility to safeguard public health by defining what constitutes acceptable indoor air quality and taking steps to ensure that those criteria are met. Examples of government actions that might be warranted include specification of minimum ventilation rates necessary to achieve healthful indoor air quality, establishment of emission limitations for building materials, and development of indoor air quality guidelines or standards for important contaminants.

ADDRESSING INDOOR AIR QUALITY ISSUES

The specter of a major public health threat from contaminated indoor air presents government decisionmakers with a dilemma. Is the problem serious enough to warrant government intervention, and if so, what preventive or remedial actions are appropriate? Unfortunately, informed policy decisions are hindered by a paucity of data on the magnitude as well as the scope of associated health risks. Before the need for government actions to protect public health can be assessed adequately, a coordinated and comprehensive effort must be undertaken to resolve critical technical uncertainties, policy issues, and regulatory questions.

The process of addressing indoor air quality issues involves five major components: problem definition, health risks estimation, mitigation measures assessment, identification and resolution of public policy issues, and decisions about appropriate government responses (see Table 1).

Table 1. Summary of the major steps in the process of addressing indoor air quality issues.

I. Problem Definition

- Emission Sources
- Dilution
- Indoor Concentrations
- Activity Patterns
- Exposures
- Health Consequences

II. Health Risk Assessment

- Number of People Exposed
- Severity of Exposure
- Dose-Response Relationship

III. Applicability of Mitigating Measures

- Ventilation
- Source Removal
- Source Modification
- Air Cleaning
- Behavioral Adjustment

IV. Resolution of Policy Issues

- Building "Publicness"
- Conservation Benefits
- Voluntary Versus Nonvoluntary Risks
- Importance of Short-Term/Long-Term Health Effects
- Public Versus Private Responsibility
- Local, State, or Federal Intervention
- Appropriate Government Responses

V. Alternative Government Responses

- No Action
- More Research
- Public Information
- Economic Incentives
- Moral Suasion
- Legal Liability
- Guidelines
- Rules/Regulations

CONCLUSION

Evidence continues to mount that air pollution is a problem indoors as well as out. The current national trend towards energy-efficient buildings has raised concerns about possible adverse effects on human health. Unfortunately, insufficient data are currently available to assess public health risks associated with energy conservation measures, such as reducing air-exchange rates, switching fuels, and adding insulation. Nevertheless, it is clear that reducing forced and natural ventilation can cause a buildup indoors of toxic and carcinogenic emissions from tobacco combustion, building materials, unvented combustion appliances, furnishings, and underlying soil. Increasing reports of building-related illness among occupants of homes and offices provide evidence of the direct relationship between the quality of indoor air and acute or short-term health effects. While less well-defined, the possibility of chronic, irreversible health consequences due to indoor exposures cannot be ignored.

Realization that indoor air quality in nonindustrial environments (e.g., residences, offices, schools, commercial and public buildings), is often less healthful than that outdoors presents policymakers with a conundrum. National Ambient Air Quality Standards have been promulgated for several outdoor air pollutants to protect public health. As a result, billions of dollars have been spent to control atmospheric emissions from mobile and stationary sources. Yet, even for those airborne contaminants that have both outdoor and indoor sources, such as carbon monoxide and nitrogen dioxide, exposures to concentrations above the standard frequently occur indoors due to indoor combustion sources. Although the health effects of breathing carbon monoxide or nitrogen dioxide are the same indoors as out, existing NAAQS apply only to air external to buildings.

If only the health implications of exposures are considered, then there is no obvious reason why air quality standards should not apply indoors as well as out. In actuality, however, the prohibitive costs and practical difficulties of monitoring and enforcing standards in tens of millions of buildings make application of the complex Clean Air Act regulations difficult, if not impossible. Furthermore, if NAAQS were extended indoors, every state would immediately be in noncompliance for carbon monoxide, nitrogen dioxide, and particulate matter, thereby necessitating enactment of specified penalties.

Indoor air pollution raises complicated public policy questions about the role of government in safeguarding health and welfare inside buildings. Although differences between indoor and outdoor environments clearly have policy ramifications, little attention has been given to identifying and resolving important issues. Nevertheless, these questions must be addressed in a systematic and comprehensive manner before real progress can be made in safeguarding indoor air quality.

Mr. SCHEUER. Thank you very much, Dr. Sexton.

Congressman Bob Smith, New Hampshire.

Mr. SMITH. Thank you, Mr. Chairman. Got the State right this time.

Dr. Stolwijk, first of all, does the language in the air pollution bill that is now in Superfund, does it provide the direction we need? Is that satisfactory to you?

Dr. STOLWIJK. The language in the Clean Air Act Amendments of 1970 were adequate for me. I think that, however, the language that is provided in the Superfund bill will be very helpful in stating very precisely that not only is there a directive to—I think the language as now stated assigns a responsibility, a specific responsibility that could be not seen in prior directives; and in that sense I feel very happy about it.

Mr. SMITH. I guess from our concern here on the committee is, have we answered legislatively what you feel you need?

Dr. STOLWIJK. I think this will be very helpful, and in the sense that constructions that say it is not the EPA's mandate will no longer be possible.

Mr. SMITH. All right. And two questions related to that, and then I'd like to ask a question of Dr. Wells.

Do we have a real indoor air quality strategy, No. 1? And No. 2, do we have the program and the funding to implement it, in your opinion?

There were some criticism earlier in the testimony regarding the fact that we didn't have a real strategy and we seem to be incoherent.

Dr. STOLWIJK. I think there was not a policy or a commitment to doing what needed to be done. I think once that policy and that commitment gets pronounced, not only here but also within the agency, then the people and the competence to carry out a comprehensive program I think are in view.

So the reason for not having a comprehensive program was basically the doubt as to whether there was a mandate or whether this kind of activity would, on a continuing basis, be permitted under the authorizations and the mandate that the agency had. I think that was the question. I think you have helped resolve that question, I think, very adequately with the language that you have introduced.

Once there is a commitment to improve indoor air quality in the Nation, then the details of the strategy will become visible I think, and become very apparent to all the people that have concerns in this area. And I think the kind of strategy that would set up a—it would sort of do a little bookkeeping as to what we know about the various pollutants because, as Dr. Sexton said, it is a whole lot of pollutants each of which is somewhat different. And once there is a set of bookkeeping that identifies for all to see what we know and what we don't know, and what needs to—and that would then specify what needs to be done, and the outcome will allow everybody to see how important it is.

And I think once that kind of information is available to legislators, then it will be easier to judge whether or not the authorizations and appropriations are adequate. But I think it would be better have that recognition come from a statement of the problem that is understandable and clear and persuasive.

Mr. **Smith**. Thank you. Mr. Chairman, may I ask a couple of questions of Dr. Wells? I heard the bell go off there.

Mr. **Scheuer**. By all means, Bob.

Mr. **Smith**. OK, thank you.

Dr. Wells—this somewhat relates to you, too, Dr. Sexton, and your question, but specifically—and your comments. Specifically, in your regard to your comments about passive smoke that—I found your study interesting. Can you—maybe you have done that in your charts. If you did, I couldn't understand it——

Mr. **Wells**. Yes.

Mr. **Smith** [continuing]. Based on your abbreviations there. But could you break that down a little bit? For example, does that mean that a person exposed to—in a 12- by 12-foot room, 5 days a week, working, to two packs of cigarettes, or one pack? Is there any way that you could give us some idea of what the risk is to come up with those kind of figures? I mean, is it being exposed to one pack of cigarettes in a 12- by 12 room, 5 days a week, 8 hours a day? Can you give me anything like that, or is that not possible?

Mr. **Wells**. Yes, I can give you an idea. Most of the data that have been collected are spouse data. In other words, these are people that have been married——

Mr. **Smith**. Not in the workplace but in the home.

Mr. **Wells** [continuing]. For a long period of time. There is very little data on workplace exposure.

On the other hand, the excess risks that I noted are for what I would call all levels of exposure; that is, to ex-smokers—you know, suppose the husband quit smoking 10 years ago. They are included in there. And it would be exposure to light smokers and heavy smokers, and so on.

If you are talking about exposure at home simply to a heavy smoker, you are talking about excess risk twice the numbers that I was talking about.

Mr. **Smith**. So are you saying that—is that one, say, one, I don't know—a cigarette an hour in, say, an area as large as a home, or even as large as a small office space?

Mr. **Wells**. Would—would be, would put you at risk.

Mr. **Smith**. Is that what you're saying?

Mr. **Wells**. The kinds of risks I'm talking about are at home. Now you don't—you're at home more than you're at work. You work 5 days a week, and you're——

Mr. **Smith**. Some of us are.

Mr. **Wells**. Yes. [Laughter.]

Well, some of us. But the people, most of the ones that made up these data bases were home more than they were at work. So my guess is the exposure at work is somewhat less dangerous, although there are some data that indicate it is more dangerous. Because if you are in a big room with several people smoking, instead of two an hour or one an hour as you were talking about you might have five an hour. So it's, you know, it's hard to tell.

But in all these carcinogenic situations there isn't any threshold risk. Every time you're exposed to this stuff it adds on——

Mr. **Smith**. Adds on.

Mr. Wells [continuing]. To whatever you've been exposed to before. So I look on the whole thing as dangerous, is my reaction.

Mr. Smith. Just one final comment, Mr. Chairman, if you'd bear my indulgence.

The, I think we're getting, all of us as Members of Congress, of course, are getting a lot of mail on this subject, especially on whether or not smoking should be eliminated in Federal, you know, in the Federal buildings specifically, and also in the workplace in general.

I guess, doesn't it boil down to the fact that if this kind of—if you were providing testimony here about something coming out of one of the vents in this room providing the kinds of toxins you're talking about in that cigarette smoke, it wouldn't be any question. But the fact is, if somebody is smoking a cigarette and supposedly, I'm a nonsmoker, so how one enjoys that I don't know. But supposedly somebody enjoys that. And because they enjoy it, therefore, that puts the pressure on us.

But in regard to what you're saying, we talk about whether in acid rain, for example, the sulfur dioxide emissions, that's something that a factory, a dirty old factory puts in the air, so that's awful and that's bad and we need to do something about acid rain. But when somebody who enjoys smoking a cigarette puts it in the air, why that somehow makes it more justifiable and more tolerable, at least to those people who smoke. But to those of us who don't smoke we feel differently. So you see what we're facing.

I mean, isn't that really a part of the problem? We're dealing with a situation here of one, somebody enjoying something, rather than—isn't that really the issue?

Mr. Wells. Well, Dr. Sexton may want to comment on that, but you're 100 percent correct, that is the issue. But I know in my personal life increasingly I avoid cigarette smoke.

Mr. Smith. I mean, we spend millions of dollars in research on acid rain, which I support—and the chairman knows that. And he does, too, support it—and also looking for more money to implement that. And, but, you know, again it's—these are dirty old factories, coal-burning utilities that are producing this. Why not apply the same set of standards? This is something we know about. It's easier to control—it should be easier to control that cigarette smoke than it is that toxin—of course, I know one's indoor and one's outdoor, but you know——

Mr. Wells. We should use the same standards.

Mr. Smith. Exactly.

Mr. Wells. Whether it's politically possible to do things about it is another question.

Maybe you want to answer that.

Dr. Sexton. Well, I would just say that, obviously, tobacco smoke has been socially sanctioned for a long time, although there're beginning to be questions, and it's much easier to point a finger at a smokestack who isn't really a person. I agree with you. And where it's someone you know, a friend who smokes, it's sometimes difficult to deal with that.

Mr. Smith. I say this with the utmost respect. And a final comment, not meant to be derogatory at all. But in a sense it really frustrates me and in a sense angers me that we have to pay for studies about cigarette smoke of the type that you're doing because I think the verdict is in, and I think it is very simply a matter of whether or not we, as a nation, certainly the Congress, and as individuals, have the guts to say no cigarette smoke. I don't think we need more studies. I think you've convinced us. You've just, you know, reiterated it, and I think that really I get to the point where I would rather spend it somewhere else, especially with these big deficits, and say, listen, let's just stop the cigarette smoking in the workplace.

Maybe I'm wrong because I'm a nonsmoker. But, thank you.

Mr. SCHEUER. Very interesting, though, really.

Let me ask you, Dr. Stolwijk, you said there was some question of whether EPA is answering the right questions. Can you tell us what questions you think we ought to be addressing them, and what questions you think they ought to be addressing themselves?

Dr. STOLWIJK. I think the first questions that you have a right to have answers to is for each of these pollutants——

Mr. SCHEUER. Can you pull that mike a little bit closer?

Dr. STOLWIJK [continuing]. For each of these pollutants what proportion of the population is exposed to what concentration. There is considerable data about that but it has not been pulled together in a way that says this is how much we know and this is what we would like—this is what we still need to know.

Similarly, we have on the whole reasonable information about what kind of consequences or what kind of diseases are associated with a lot of pollutants. Where we are sadly lacking is in the quantitative relationships between these two. As a consequence, the advice that we can give is limited by saying this kind of activity like passive cigarette smoke causes excess something or other. The difficulty is that unless you can state it more precisely you lack what is the apparent certainty that, let's say, came out of the Surgeon General's report about smoking in general. There was a considerable persuasiveness to the relationships that could be pointed out there. We at the moment do not have this.

The other thing that is important to know, that all of this work will always be done in an atmosphere of limited resources.

Mr. SCHEUER. Very good.

Dr. STOLWIJK. Because the situation is so complex and has so many facets to it, it is important to know, to have some ordering of the priorities as to where these limited resources are going to be applied, and that I think needs to be able to be—the reasoning behind this priority allocation I think needs to be understood by all the participants before it really will be effective.

Mr. SCHEUER. I wonder if you would be kind enough to give us a list of questions which you think that we ought to make sure that the EPA addresses.

Dr. STOLWIJK. I would be happy to send you a list of questions in the mail if you——

Mr. SCHEUER. Good. That will be swell.

OK. Dr. Sexton, you indicated that there was some doubt in your mind as to whether EPA was addressing the right public policy questions in formulating their research program. Is that correct?

Dr. SEXTON. I think that there are a lot of facets that need to be taken into account when they are designed.

Mr. SCHEUER. A lot of what?

Dr. SEXTON. A lot of facets that need to be taken into account when they design a research program. And it is not only the issue of exposure and health effects in my mind that should come into play. There is also the issue of source apportionment. The idea where does the pollutant come from. Is it coming from an indoor source or an outdoor source or both, and what is the relative contribution?

It is also important I think to know something about time/activity patterns. Just because we know the concentration inside a building doesn't mean that there are people there, or we don't know how much time they spend there. We also don't know, as far as the sensitive subgroups, how much time they might spend in specific locations.

I think that all these things, this whole issue of exposure to both indoor and outdoor pollution has to be taken into account when they design a program to examine these questions.

Mr. SCHEUER. Well, didn't you specifically mention public policy questions, broader issues of public policy?

Dr. SEXTON. Yes. And as an example——

Mr. SCHEUER. Well, what did you have in mind when you mentioned that?

Dr. SEXTON. What I'm thinking of there is that it seems to me if we decide, for instance, that the air quality inside a person's home is, in fact, a personal choice issue, similar to what we've done for tobacco smoking or for saccharin, then the obvious idea here would be to give them information to make their own decisions.

Mr. SCHEUER. Correct.

Dr. SEXTON. If that's the case, we may not need as detailed information on health effects as we do if the Government is going to take some more direct action and set a regulation. So the level of evidence that's needed to take these various actions it seems to me is different.

Mr. SCHEUER. Absolutely right. We don't have to tell them, accurate to a gnat's eyelash, at what exact degree we pass from one level of risk to another. We can simply say this bloody business is dangerous to your health, so take reasonable remedial steps to cut it out. Correct?

Dr. SEXTON. Yes.

Mr. SCHEUER. Perhaps you'd give us a list of the public policy questions that you think ought to underline our mission as we have stated to EPA and any specific programmatic goals that you think EPA ought to be addressing. Could you submit that to us?

Dr. SEXTON. Certainly. Yes, sir.

[Copy of questions follow:]

INDOOR AIR QUALITY: IMPORTANT PUBLIC POLICY QUESTIONS

Does the role of government depend on the degree of "publicness" for a particular building? And if so, what is an appropriate response to air quality problems in private, as opposed to public, buildings?

Is consideration of the difference between voluntary and nonvoluntary risks important for choices about government intrervention?

What are the trade-offs (e.g., costs versus benefits) between energy conservation measures and healthful indoor air quality?

Should the emphasis be placed on protecting building occupants from long-term chronic exposures or short-term peak exposures?

Should everyone, including sensitive individuals (e.g., infants, the elderly, those with asthma and emphysema), be afforded the same degree of protection from indoor air contaminants?

What are the responsibilities incumbent upon different sectors of society (e.g., individuals, building owners and operators, architects, developers, contractors, manufacturers, government), which have a stake in the issue?

If it is decided that government intervention is justified, what forms of intervention are appropriate?

If government actions to ameliorate problems are deemed necessary, at what level of government (i.e., local, state, or Federal) should authority and repsonsibility be vested?

Mr. **Scheuer**. Now let me ask Dr. Wells: I have two bills in. We're doing some detailed research to find out if my colleague Bob Smith is on these bills as sponsor. One of them would prohibit smoking in all Federal buildings, and the other would prohibit smoking in all commercial airlines. Now we provide an out for the Federal buildings. We direct that the Administrator of the GSA that runs Federal buildings should negotiate with the public employee unions to set aside a special smoking place, if there is a felt need among the employees to do that, that wouldn't bother the nonsmokers, wouldn't endanger their health, and would provide a place where the smokers can endanger their own health in comfort.

Now there is also another bill pending in the Congress that would prohibit smoking on all flights of up to 2 hours. All commercial flights up to 2 hours. The assumption is that the rate of addiction and the intensity of the addiction is so appalling for smokers that we can ask them to cease and desist for 2 hours, but above and beyond that they would absolutely go berserk and attack the pilot and the stewardess and their fellow passengers and go off the wall. That we can't subject them to that amount of stress and strain.

Now we haven't proved very definitively so far as airlines are concerned, or indeed as far as Federal buildings are concerned, because the conditions vary so widely—the size of the room, the condition of the ventilator, the number of people who work there—I mean, the number of windows. I mean, there is an infinity of conditions it would be impossible to research. It seems to me it would be impossible to come up, without exorbitant expense and without doing a detailed, agonizing analysis of every single Federal facility in the country, and indeed every airline or every type of aircraft because their size varies, the length of the flight varies, the condition of the air-conditioning equipment varies, its degree of good repair varies, its effectiveness varies.

No. 1, give me your absolutely objective reaction to the wisdom of these two prohibitions, Federal buildings and commercial airline flights, all of them, 2 hours more, 2 hours less—all of them. The passenger that would absolutely go off the wall screaming and clawing would have to stay off of commercial airline flights. In your view, what is the wisdom of that? How do you weigh the societal benefits against the societal costs? How much consideration do we give to this potentially berserk smoker on a commercial airline? How much consideration do we give to the Federal employee in a Federal building who just absolutely needs his or her fix? What kind of an obligation do we have to them to provide a place, or can we say you can always go outdoors, even when it's snowing or sleeting?

How do we weigh those public policy questions? And then tell us what do you think as a matter of just pure commonsense and horse sense, to what degree of exactitude do we have to define the risk of passive smoking? To a 10th decimal point or the 50th decimal point, in order to justify a prohibition on smoking in Government buildings and on commercial airlines?

Mr. WELLS. Well, let me comment——

Mr. SCHEUER. And I address this to a person who is known for his impartiality and balance in approaching these questions.

Mr. WELLS. Well, first let's talk about addiction. I think it is true that cigarettes and nicotine are extremely addictive. If you talk to the National Institute of Drug Abuse, they'll tell you it's as addictive as cocaine is, or heroin. And it is a very difficult addiction to shake, and we've got to be kind to the smokers in that sense. I think we have to recognize they are in a terrible fix.

On the other hand, there are many institutions in this country where they have gone smoke free. The Harvard School of Public Health has just gone smoke free. I think you either have to go outside to smoke or there's some little place where you can smoke. The Blue Cross in Wilmington, DE, has gone smoke free as of last year sometime. And these organizations that do this get along fine. They don't have any trouble.

Mr. SCHEUER. I might add that there are about 300 municipalities and I think 30 or 35 States who—which have already prohibited smoking. And at the State and the city level they want to prohibit smoking in Federal buildings, many of them, but they simply don't have the jurisdiction.

Mr. WELLS. So I would say, sir, that, although it is a very addictive thing, the difficulties have been vastly overblown as to prohibiting smoking in Federal buildings or any kind of building or a workplace. So that's point No. 1.

Point No. 2 is that people who think they have not been exposed to environmental tobacco smoke still have appreciable amounts of cotinine and nicotine in their urine, in their blood plasma, and body fluids. The figures I have given you here for passive smokers, what they find is that the people that think they haven't been exposed have about one-third the evidence of passive smoking in their bodies that the people who think they have been exposed have. In other words, there may be an exposure here for the so-called true nonsmokers that's more than they think it is.

So I think if you're going to have smoking in one part of a room and no smoking in another part, or smoking in one part of a building and not in another part of the building, you're still probably exposing those nonsmokers more than you think, and more than we would allow if it were a normal carcinogenic air pollutant.

Mr. SCHEUER. You mean because the smoke-polluted air is getting into the circulation system——

Mr. WELLS. Somehow. Because——

Mr. SCHEUER [continuing]. For the building?

Mr. WELLS [continuing]. Time and again when they try to measure the body fluids of people who think they've never been exposed to tobacco smoke, they keep picking up this nicotine and cotinine, and the only source for those materials is tobacco smoke.

Mr. SCHEUER. Fascinating. Dr. Stolwijk, has EPA been providing to all of us the right kind of information to respond to the radon problem? And are they devoting adequate resources to giving us the information we need to respond to the radon problem?

Dr. STOLWIJK. I think that the best judges of that are State departments of health. They are the institutions that are on the front line of the questions that result from publicity and from what people read in newspapers and what they see on their television news.

They tell me that the number of inquiries in most States is going up dramatically; has been going up and is still going up. They also tell me that the material that is now being made available by EPA is reasonably effective in answering the questions. What they seem to feel is that after they have mailed out or given out these brochures that the public still wants to have local assurance, by local officials, as to how it applies to their particular situation or their particular condition.

They, of course, use the existing—pre-existing level of radon as a guideline, as to—as an action level, which came from another set of problems that isn't of prime concern at the moment anymore. I think that what probably—what was found in other countries, what was found to be useful in Sweden, for instance, was another set of observations also administered by local health departments. It seems to be very important that the local health departments who are on the front line be helped in dealing with the problem.

What the Swedes have done is to use a slightly higher as a level of no concern, about 6 picocuries, and the Nordic countries have then also adopted a level of about 20 picocuries per liter as an action level. That is a system that has been in operation there now for sometime, and that combined with the low-interest loans for people that want to devote some resources to their houses appears to be a working situation which in Sweden, as far as I can tell, has reduced the level of anxiety on the part of the public back to more reasonable levels.

So I think that the material is certainly helpful.

Mr. SCHEUER. Now, that most certainly is radon? You are speaking of radon and other areas.

Dr. STOLWIJK. Yes. I'm speaking specifically about radon. I'm not aware of any widespread material that EPA has available to deal with other problems.

Mr. SCHEUER. Well, let me just ask. Sweden is obviously very much—very sensitive about the problem of air pollution. How does Sweden treat the problem of smoking in public buildings and on commercial air flights?

Dr. STOLWIJK. They have a larger number of buildings that is smoke free than we do, but it is not done by regulation. It is done by adopting the local jurisdiction; the building manager, or whatever, does it. The process that you——

Mr. SCHEUER. Are the tenants, the users of the air, the breathers of the air, involved in that decision? Is there a vote of——

Dr. STOLWIJK. There usually——

Mr. SCHEUER [continuing]. The public employees, for example, in a building?

Dr. STOLWIJK. I think there usually is. I have exactly the same experience. In my building—for instance, at my department at Harvard—has had to consider what to do, and we have basically adopted the stance that all public spaces, and that means classrooms and everything else, are off-limits for smoking, and we have not seen people crawling up walls yet.

Mr. SCHEUER. Yes. Yes, Dr. Sexton?

Dr. SEXTON. I just wanted to make one statement having to do with this idea about providing people with information. I think it's appropriate, and I think it's a first step. But as far as I'm aware, there's really no program to evaluate the value of this information in improving people's choices about their indoor air quality.

It seems to me that if we're going to give people the information it would just make sense to follow up to see if, in fact, they're taking any action based on the information. And if they're not, perhaps we need to do something else. The risks of radon, if you live in a home with high levels and you've lived there for many years, is obvious and can be very high. If your parents have chosen not to take any action, it really is not your fault.

And there it might be that information is not as effective as we think it's going to be. I think it's something that we should consider looking into.

Mr. SCHEUER. Yes?

Mr. MEYERS. I just wanted to say that we do have a couple of programs—the brochures that I gave you, to find out if, indeed, people are using them and whether or not they are effective.

Mr. SCHEUER. Very good.

Dr. STOLWIJK. There is one other comment, Mr. Chairman, that I would like to make. And, we have not attached very much importance at this particular hearing to the large office buildings and what its fate is. Currently NIOSH has taken on the jurisdiction to look at them, but I think not terribly effectively because they're looking at other kinds of workplaces. But I might point out that many of us in a country that is going more and more in the direction of and employing more and more people in service industries, the number of people that are exposed for 8 hours a day to—in their particular work environments where they expect the quality that is equivalent to the residential quality and where they may not be getting it, there is a problem there to be resolved that at the moment is not being addressed very eagerly by anybody.

And organizations like the American Society for Heating, Refrigerating & Air-Conditioning Engineers, who are currently charged with the development of ventilation standards for the whole country, because they adopt that responsibility, are finding that there is very little in the way of help. Other than some research that goes on in the Department of Energy, there is very little help that they can count on in making recommendations in their standards which ultimately get adopted by reference or directly into most building codes. And here is a private organization trying to cope because nobody else is. And it is a very important area that I think deserves more, more support in terms of research and advice from the Federal Government than they're currently getting.

Mr. SCHEUER. Dr. Stolwijk, EPA has proposed to conduct a national multipollutant study, a field study of combustion gases and volatile organic compounds, as their major indoor air pollution initiative. In your opinion, would such a study, such a survey provide useful information?

Dr. STOLWIJK. It would clearly always provide useful information. I would be hesitant to recommend that they commit to such a study on the scale that is being discussed if they do not prior to that commitment evaluate completely what is already known and what is likely to be learned in addition from the evaluation of that which is already known. And I think the hesitancy in the panel endorsing that particular study really came from the fact that it was a completely de novo enterprise that was not building on what was already known and, as a result, risked committing scarce resources to areas that didn't really need it.

Mr. SCHEUER. Or reinventing the wheel and studying areas that have already been studied and restudied.

Dr. STOLWIJK. We all like to reinvent the wheel from time to time.

Mr. SCHEUER. Yes. Let me ask all of you, all too often the public pays attention only to cancer risks. What other health outputs or endpoints, or whatever, should be of concern to public health officials? What do we know about the relationship between indoor air pollutants and health results other than cancer; in other words, respiratory diseases, neurotoxic effects? Anybody.

Mr. WELLS. I guess I commented earlier that there are heart effects showing up in the passive smoking literature. Those are very early studies. It would be nice to have more. But they are showing up.

And there is some evidence of emphysema and chronic bronchitis in the work that Hariyama did in Japan. But the nonsmoker death rates for those diseases are so low that probably it's not an area that's all that fruitful. But certainly it would be worth pursuing more work on the heart effects.

And then there are, of course, people that are rather allergic, or think they are, to tobacco smoke; and when they try to work in a smoky workplace, it's extremely painful for them. And that's also true on aircraft.

I had an instance up at Harvard here the other day where the wife of an old dean of mine had to get up and leave the dining room because two people lit cigarettes behind her in the dining room. She just happens to be allergic to cigarette smoke. But she didn't say anything, she just left. In a workplace, you can't very well leave.

So, there are asthmatic effects, all sorts of things like that, but many of those are pretty well known already.

Dr. STOLWIJK. Mr. Chairman, I might add to that, that in addition to that, the serious consequences or the really serious acute and disabling consequences, there are effects on the immunity to respiratory diseases, to mild respiratory disease that are quite severe and that clearly have a relationship to the level of indoor air pollutants, or the level of outdoor air pollutants, for that matter; and if we consider that we have, all of us, something in the order of two to four head colds or respiratory infections per year that to some extent disable us, and if we see that in our—if we see that under conditions where this has been measured that there is a clear increase in the incidence of such events occurs when air pollutants go above a certain level, it then becomes visible. That there is a national impact on total days of restricted activity that might occur, which is in the hundreds of millions of days that people aren't working as well as they could because they have these various not completely incapacitating illnesses.

Similarly, there is especially in the office environment a consequence of the exposure to the combined organics and other things that might be in the air which clearly lead people to work at a reduced work capacity, partially because of being concerned or partially because of being uncomfortable. And the loss of that work capacity also, if it were properly known and could properly be assessed, would indicate a very high effect of relatively mild levels of indoor—of lacks of indoor air quality or indoor air pollutants that in the national frame represent a cost, a loss of productivity that is difficult to measure but I feel certain is quite substantial.

And at the moment it is not really anybody's business to look into that, and no individual employer has sufficient incentive to actually investigate that or look after it. I believe that the research that leads to those recognitions and to knowing what to do about them clearly is a responsibility of the Federal Government.

Mr. **Scheuer**. Then you think it hasn't been accurately described or measured, but you feel that it's real and it does provide some reduction of effectiveness in the workplace?

Dr. **Stolwijk**. I think anytime that I get called into a troubled building one thing I do notice is that the level of productivity of the workers in that building is severely affected.

Mr. **Scheuer**. Yes.

Dr. **Stolwijk**. And that usually gets the attention of the managers or the people that run that enterprise, and it is only when it becomes so significant that it begins to bother that something gets done about correcting it.

Mr. **Scheuer**. And that's why they call you in?

Dr. **Stolwijk**. It seems to be.

Mr. **Scheuer**. Yes. Are the noncancer effects of indoor air pollution likely to be more important from a public health perspective than cancer, as far as indoor air pollution is concerned?

Dr. **Stolwijk**. I find it very difficult to weigh such things against one another. I know what will get more public attention, and that will be a thousand cancer deaths per year which can be assigned or which can be attributed to exposures of this type or others that we can't quite identify—getting a lot more attention than 20 million getting an excess cold.

Mr. **Scheuer**. Than 20 million people getting?

Dr. **Stolwijk**. Getting an excess cold.

Mr. **Scheuer**. Yes. All right, my last question. What are the major knowledge gaps with respect to what we know or don't know on indoor air pollution? Anybody.

Dr. **Sexton**. Mr. Chairman, I think that that's very pollutant specific, and it also tends to be microenvironment specific. By that I mean——

Mr. **Scheuer**. Could you translate——

Dr. Sexton. Sure.

Mr. Scheuer [continuing]. That into English——

Dr. Sexton. Sure.

Mr. Scheuer [continuing]. For the radio and television audience?

Dr. Sexton. It also tends to be specific by pollutant and by the environment that we're talking about. For instance, we know a substantial amount in terms of the levels of nitrogen dioxide in residences. We don't know as much as we might, but we know quite a bit about that particular pollutant in that environment. We don't know very much about any pollutants in the office environment. We know, are beginning to know more about radon in the home environment. So in terms of answering that kind of a question, you really would have to deal with each pollutant specifically and each environment specifically.

Mr. Scheuer. Anybody else?

Mr. Wells. I guess I'd like to make a comment.

Mr. Scheuer. Yes.

Mr. Wells. That I think we've, in indoor air pollution we may have been preoccupied with lung cancer, and we might want to look more broadly into cancers other than lung and cardiovascular effects if we're thinking about long-term chronic problems. And then the kind of thing Dr. Stolwijk was talking about, acute problems and productivity, I think there's room for work there also.

Dr. Stolwijk. I think that as a general class for most pollutants the kind of question that one gets asked very often is what is the level that I should be at? Is there a guideline of some kind? And that guideline then translates into what is the level of effect at that concentration?

The ability to answer that question at the moment is much less than our ability to say, yes, there is a connection between this exposure and that effect. But having said that, the next question that a consumer or the public will ask is, how bad is it? And that how bad is it is something that we are very poorly informed about. That is for most of the areas of concern is I think generically the weakest link in the decision chain.

Mr. Scheuer. Dr. Wells, you mentioned before that you had aggregated the results from a number of surveys and sort of pulled them together. Have they been published?

Mr. Wells. The aggregation that I have made was presented, as I said, at the Minneapolis meeting of the Air Pollution Control Association and will be published in the proceedings of that meeting.

Mr. Scheuer. Well, I wonder whether you'd be willing for us to publish them as part of this record?

Mr. Wells. You're certainly free to publish that paper if you want to.

Mr. Scheuer. All right. If you'll give us the paper, we'll append——

Mr. Wells. I appended it to my remarks.

Mr. Scheuer. Oh, very good.

Mr. Wells. Yes.

Mr. Scheuer. You appended the paper that you gave——

Mr. Wells. That's right.

Mr. Scheuer [continuing]. Minneapolis? Very good.

Mr. **Wells**. Well, I updated it a little bit to make the tables a little more clear.

Mr. **Scheuer**. Excellent.

Mr. **Wells**. But it's basically the same paper.

Mr. **Scheuer**. Excellent. Well, this has been an extremely interesting panel. You've been patient, long suffering. You've been very thoughtful and stimulating. We thank you very, very much.

The meeting is adjourned at the call of the Chair.

[Whereupon, at 3:38 p.m., the subcommittee was adjourned, to reconvene subject to the call of the Chair.]

APPENDIX

UNITED STATES HOUSE OF REPRESENTATIVES

NEWS RELEASE

CONGRESSWOMAN CLAUDINE SCHNEIDER
SECOND DISTRICT–RHODE ISLAND

1512 LONGWORTH BUILDING, WASHINGTON, D.C. 20515

For Immediate Release

Contact: Liz Raisbeck
202/225-2735

SCHNEIDER CALLS FOR NEW FEDERAL STRATEGY ON INDOOR AIR POLLUTION

WASHINGTON: Representative Claudine Schneider called on the Environmental Protection Agency to move immediately to establish a coordinated federal research strategy on indoor air pollution.

"EPA has had an indoor air research program since 1983, and it still has not created a clear set of goals and strategies for the program," Schneider said. "Both time and money are short. We cannot afford to dawdle one extra day on this program.The states are greatly in need of leadership and technical assistance so that the risks can be identified and eliminated," she said at a hearing today before the Subcommittee on Natural Resources on the indoor air research program. She criticized EPA for spending many months developing a very expensive survey instead of developing a research plan.

Air pollutants such as radon, tobacco smoke, formaldehyde and asbestos have been found in concentrations significantly higher in indoor than in outdoor air. Since the average American spends 70% of his/her time indoors, indoor air pollutants may represent a significant threat to human health. EPA recently estimated that elevated levels of radon may be responsible for as many as 20,000 lung cancer deaths every year.

(over)

Indoor air pollution could be a serious problem in Rhode Island. The state's program for monitoring asbestos in schools has determined that 90% of the areas in schools surveyed so far contain asbestos, and 25% of the areas surveyed pose such a health threat that federal funds have been made available to clean up the problem. Over 20% of the 300 Rhode Island houses surveyed for formaldehyde contained levels which pose a threat of respiratory problems to inhabitants of houses. The state has only just begun to survey Rhode Island homes for potential radon problems.

Last year Congresswoman Schneider introduced a bill requiring EPA to establish a comprehensive indoor air quality research, monitoring and mitigation program. This bill was incorporated into the Superfund program agreed to by the House and Senate last month.

AIR POLLUTION CONTROL ASSOCIATION'S
KEYNOTE ADDRESS FOR THE INDOOR
AIR PLENARY SESSION
DONALD J. EHRETH
ACTING ASSISTANT ADMINISTRATOR
FOR RESEARCH AND DEVELOPMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
JUNE 23, 1986

GOOD MORNING,

IT IS TRULY AN HONOR AND A PLEASURE TO SPEAK TO THE GROUP ASSEMBLED HERE TODAY. YOU, THE PROFESSIONALS, THE AIR POLLUTION EXPERTS, PROBABLY DO NOT STOP OFTEN ENOUGH TO TAKE CREDIT FOR THE AMOUNT OF PROGRESS YOU HAVE MADE IN THE PAST TEN TO FIFTEEN YEARS. GLANCING AT THE THEMES OF THIS CONFERENCE, ONE IS IMMEDIATELY STRUCK BY THE FACT THAT WE ARE BEGINNING TO DEAL WITH A SET OF PROBLEMS AND PRIORITIES TODAY DIFFERENT FROM THOSE OF TEN TO FIFTEEN YEARS AGO. MAYBE THIS IS WHERE LIFE BEGINS.

I CAN'T HELP BUT BE REMINDED OF A STORY THAT MY FORMER BOSS BERNIE GOLDSTEIN USED TO SHARE WITH ME WHEN WE TALKED ABOUT THE WAY THE ENVIRONMENTAL PROGRAM HAS CHANGED OVER THE PAST SEVERAL YEARS. THERE WERE THREE MEN OF THE ROBE: A PRIEST, A METHODIST MINISTER AND A RABBI THEY WERE SEATED AT A RESTAURANT DISCUSSING WHEN LIFE BEGINS. OBVIOUSLY, WHEN YOU HAVE A GROUP SUCH AS THIS DISCUSSING THIS ISSUE, THE DISCUSSION CAN GET RATHER HEAVY. THE CATHOLIC PRIEST, NOT UNEXPECTEDLY, STATED WITH CONVICTION THAT LIFE BEGINS AT CONCEPTION AND CERTAINLY ANYTHING TO IMPAIR THE LIFE OF THAT NEWLY DEVELOPED SOUL WOULD BE ENDING LIFE. THE METHODIST MINISTER HAD A SLIGHTLY DIFFERENT IMPRESSION. HE MADE THE POINT THAT LIFE BEGAN AT THE TIME OF BIRTH. THE RABBI LEANED BACK AND GAVE HIS ANSWER SEVERAL MINUTES OF DEEP THOUGHT BEFORE HE CALMLY ENTERED THE CONVERSATION. POINTING HIS FINGER IN THE AIR, HE SAID, "NO, GENTLEMEN AND MY GOOD FRIENDS, I MUST DISAGREE! LIFE MUST CERTAINLY BEGIN WHEN THE DOG DIES AND THE CHILDREN LEAVE HOME."

WELL, PERHAPS THE "CHILDREN" HAVE BEEN THE PROBLEMS THAT WE FACED IN THE LAST TEN TO FIFTEEN YEARS. TO SOME EXTENT WE CAN SAY THAT WE'VE MOVED BEYOND THOSE PROBLEMS. ON THE OTHER HAND, WE CAN'T SAY THAT THE DOG HAS DIED OR THAT POLLUTION IS NO LONGER A PUBLIC ISSUE. IN THE EARLY TO LATE 70's, WE LITERALLY USED BULL-DOZERS, CONCRETE AND STEEL, AND TODAY THE AIR IS CLEANER; THE SAME IS TRUE FOR OUR WATER. THE ANSWER WAS A TECHNOLOGY BASED APPROACH TO REDUCING ENVIRONMENTAL POLLUTION. WITH MUCH OF THE POLLUTION CONTROL MEASURES IN PLACE, WE NOW ARE EVALUATING ADDITIONAL CONTROLS ON THE BASIS OF RISK AND RISK REDUCTION.

LOOKING OVER THE AGENDA OF THIS CONFERENCE, ONE IMMEDIATELY SEES THAT THERE ARE MANY SESSIONS DEDICATED TO THE CONVENTIONAL AIR POLLUTION PROBLEMS. COULD YOU EVER IMAGINE AN APCA CONFERENCE WITHOUT A SESSION ON PM-10 MONITORING? I'M SURE THAT SESSION WILL BE JAM-PACKED. WHAT APCA CONFERENCE WOULDN'T ADDRESS FLUE GAS DESULFURIZATION WITH ACID RAIN LEGISLATION NOW WINDING ITS WAY THROUGH CONGRESS.

BUT WHO WOULD HAVE IMAGINED TEN TO FIFTEEN YEARS AGO THAT AN APCA CONFERENCE WOULD HAVE A SIGNIFICANT PORTION OF ITS AGENDA DEDICATED TO HAZARDOUS WASTE MANAGEMENT? TEN TO FIFTEEN YEARS AGO, HAZARDOUS WASTE PROBABLY WASN'T A STAPLE IN OUR AIR VOCABULARY. NO ONE GAVE MUCH ATTENTION TO THE AIR EMISSIONS COMING OUT OF THE HAZARDOUS WASTE LANDFILLS OR WHERE THE CONSTITUENTS OF THOSE LANDFILLS WERE GOING. NO ONE GAVE TOO MUCH ATTENTION TO WHAT POLLUTANTS MIGHT BE FORMED DURING THE COMBUSTION PROCESS. NOW WE HAVE A NEW VOCABULARY FILLED WITH TERMS SUCH AS "PIC's" AND "POHC's".

THIS YEAR APCA HAS BEEN CONDUCTING REGIONAL SEMINARS THROUGHOUT THE COUNTRY AIMED AT IMPROVING THE STATE OF KNOWLEDGE OF ANOTHER RELATIVELY NEW ISSUE OR IN THE MINDS OF SOME PEOPLE, NOT SO NEW. THE EXPOSURES TO A NUMBER OF POLLUTANTS FOUND INDOORS SEEM TO BE SIGNIFICANTLY GREATER THAN EXPOSURES TO THE SAME SET OF POLLUTANTS FOUND IN THE OUTDOOR ENVIRONMENT. THIS RAISES QUESTIONS OF RISK OR RELATIVE RISKS. IN CONCERT WITH THE RISK BASED APPROACH ADOPTED BY EPA IN THE EARLY '80's, WE ARE BEGINNING TO ASK WHAT ARE THE SOURCES OF HEALTH RISKS? IS INDOOR AIR MORE OR LESS IMPORTANT AS A SOURCE OF THE RISKS THAN OUTDOORS? HOW DO WE DEAL WITH THEM?

THERE HAS BEEN A GROUP OF DEDICATED SCIENTISTS AND PUBLIC INTEREST ORGANIZATIONS THAT HAVE DOGGEDLY DEMONSTRATED THEIR CONCERNS ABOUT INDOOR AIR. IT HAS BECOME ONE OF THE MOST EMOTIONAL ISSUES THAT I HAVE EVER DEALT WITH IN MY FOURTEEN PLUS YEARS AT THE ENVIRONMENTAL PROTECTION AGENCY. AND THIS YEAR INDOOR AIR POLLUTION IS A FOCAL POINT OF APCA's CONFERENCE. IT IS AN ISSUE WHICH IS NOW RECEIVING ATTENTION FROM CAPITOL HILL, CONSUMER GROUPS, PRIVATE SECTOR GROUPS, ACADEMIA, FEDERAL AGENCIES, STATE GOVERNMENTS, AND LOCAL GOVERNMENTS.

THERE IS GROWING PRESSURE FOR THE FEDERAL GOVERNMENT TO TAKE A LEADERSHIP ROLE IN ADDRESSING THIS PROBLEM. IN 1982, THE AIR POLLUTION CONTROL ASSOCIATION PASSED A RESOLUTION URGING THAT THE CLEAN AIR ACT BE AMENDED TO AUTHORIZE AN INDOOR AIR PROGRAM. A RESOLUTION FROM THE NATIONAL GOVERNORS ASSOCIATION (NGA) ALSO CALLED FOR A STRONGER FEDERAL PRESENCE IN INDOOR AIR. PRIVATE SECTOR GROUPS, LIKE THE CONSUMER FEDERATION OF AMERICA, WERE MAKING INDOOR AIR QUALITY A HIGH PRIORITY CONCERN.

FOR TWO YEARS, INDOOR AIR HAS BEEN THE THEME OF THE CLEAN AIR WEEK SPONSORED BY THE AMERICAN LUNG ASSOCIATION. THE GENERAL ACCOUNTING OFFICE HAS CALLED FOR A GOVERNMENT-WIDE COORDINATION EFFORT IN INDOOR AIR AND FOR REVISION OF THE CAA TO PERMIT REMEDIATION OF INDOOR AIR QUALITY PROBLEMS.

THE CONGRESS IN EACH OF FISCAL YEARS 1984, 1985, AND 1986, HAS SPECIFICALLY APPROPRIATED FUNDING FOR EPA RESEARCH IN INDOOR AIR QUALITY. IN ADDITION, LEGISLATION HAS BEEN INTRODUCED IN BOTH THE HOUSE AND SENATE WHICH WOULD AUTHORIZE EPA TO CONDUCT RESEARCH AND INFORMATION DISSEMINATION ACTIVITIES ON INDOOR AIR ISSUES.

INDOOR AIR POLLUTION AT EPA IS NOT A REGULATORY PROGRAM. WHY THEN IS IT RECEIVING SO MUCH ATTENTION? WHY DOES IT RECEIVE THIS HIGH DEGREE OF ATTENTION BY SUCH A BROAD SEGMENT OF OUR POPULATION? I BELIEVE ITS BECAUSE WHAT WE'RE REALLY CONCERNED ABOUT IS UNDERSTANDING THE TOTAL RISKS THAT WE ARE EXPOSED TO IN OUR DAY-TO-DAY LIFE. QUANTIFYING THE RELATIVE RISKS OF EXPOSURE WE EXPERIENCE FROM AN INDOOR SETTING VS. THE OUTDOOR SETTING IS IMPERATIVE. WITHOUT THAT KNOWLEDGE WE ARE NEVER GOING TO BE SURE THAT WE HAVE EMBARKED UPON THE MOST COST EFFECTIVE OR THE MOST EFFECTIVE RISK REDUCTION STRATEGIES. WITH BETTER EXPOSURE DATA, WE CAN MORE ACCURATELY ASSESS THE RISKS OF THE POLLUTANTS WE ENCOUNTER DAILY. WHY ARE WE CONCERNED?

AS I SAID, MANY TOXIC SUBSTANCES FOUND IN THE AMBIENT AIR MAY BE FOUND INSIDE HOMES AND BUILDINGS. MUCH CONCERN IS NOW FOCUSED ON THE NON-INDUSTRIAL WORKPLACE, SUCH AS THE "SICK BUILDING SYNDROME," IN WHICH VARIOUS HEALTH SYMPTOMS SUCH AS IRRITATION OF MUCOUS MEMBRANES, HEADACHES, DIZZINESS, AND NAUSEA, HAVE BEEN DOCUMENTED. ACCORDING TO A 1979 RAND CORPORATION STUDY, AN ESTIMATED 25% OF THE U.S. WORKFORCE CAN BE FOUND IN OFFICES AND OTHER NON-INDUSTRIAL WORK SITES. A STUDY REPORTED BY THE AMERICAN SOCIETY OF HEATING, REFRIGERATION, AND AIR CONDITIONING ENGINEERS (ASHRAE) IN 1982 INDICATED THAT, IN MANY SITUATIONS, OUTSIDE AIR DAMPERS TO BUILDINGS ARE BEING CLOSED OFF BY BUILDING OWNERS AND OPERATORS IN THE INTERESTS OF ENERGY CONSERVATION. THESE DAMPERS REPLACE THE AIR USED UP THROUGH HUMAN RESPIRATION AND OTHER ACTIVITIES, BUT, WHEN CLOSED, SIGNIFICANTLY REDUCE THE IN-FLOW OF OUTSIDE AIR, THEREBY ALLOWING ANY TOXIC MATERIALS PRODUCED IN THE BUILDING TO INCREASE IN CONCENTRATION AND INCREASE THE POTENTIAL HEALTH RISK TO THE BUILDING OCCUPANTS. LONG TERM HEALTH IMPACTS ARE GENERALLY NOT KNOWN AT THIS TIME. IN SOME CASES, HOWEVER, ACUTE EXPOSURES HAVE LED TO ILLNESSES AND EVEN DEATHS, AS IN THE CASE OF LEGIONNAIRES DISEASE.

HOMES ARE OFTEN SIGNIFICANT SOURCES OF HEALTH RISKS DUE TO CONTAMINATION BY RADON FROM SOIL GAS, FROM TERMITICIDES, VOLATIZATION OF FORMALDEHYDE IN BUILDING MATERIALS, COMMERCIAL CLEANING PRODUCTS, AND COMBUSTION PRODUCTS FROM COOKSTOVES, SPACE HEATERS, WOODSTOVES, AND SMOKING. EPA's TEAM STUDY OF VOLATILE ORGANIC COMPOUNDS (VOC) IN RESIDENCES HAVE SHOWN THAT INDOOR CONCENTRATIONS OF KNOWN CARCINOGENS, LIKE BENZENE, CHLOROFORM, AND TETRACHLORETHYLENE, EXCEED OUTDOOR CONCENTRATIONS BY FACTORS OF 2 TO 10.

LEVELS OF INDOOR RESPIRABLE PARTICLES PRODUCED BY TOBACCO SMOKE IN BOTH COMMERCIAL BUILDINGS AND HOMES HAVE BEEN OBSERVED TO EXCEED OUTDOOR CONCENTRATIONS BY FACTORS OF 2 TO 30. RECENT SURVEYS OF RADON IN HOMES HAVE SHOWN SIMILAR RATIOS OF INDOOR TO OUTDOOR CONCENTRATIONS. RADON LEVELS IN THE READING PRONG REGION OF PENNSYLVANIA, WHICH HAVE GOTTEN SUBSTANTIAL NATIONAL ATTENTION, HAVE BEEN OBSERVED TO BE 1,000 TIMES HIGHER INSIDE THE HOME THAN OUTSIDE IN THE AMBIENT AIR. OTHER STUDIES HAVE SUGGESTED THAT CARBON MONOXIDE MAY BE A PROBLEM IN SOME BUILDINGS DUE TO SEEPAGE OF THE CO FROM MOBILE SOURCES IN ADJACENT OR UNDERGROUND GARAGES. THE FULL SIGNIFICANCE OF HIGHER INDOOR LEVELS OF THESE CHEMICALS HAS NOT BEEN DETERMINED; HOWEVER, IF OUTDOOR LEVELS OF THE SAME SPECIES HAVE BEEN SHOWN TO PRODUCE SIGNIFICANT HEALTH RISKS, THEN IT FOLLOWS THAT INDOOR RISKS MAY BE THE SAME OR HIGHER. ACCORDING TO THE ESTIMATE IN 1975, FORMALDEHYDE CONCENTRATIONS IN U.S. RESIDENCES MAY EXCEED LEVELS PERMITTED FOR CONTINUOUS OUTDOOR EXPOSURE AS REGULATED IN SEVERAL EUROPEAN COUNTRIES AND EXCEED LEVELS PERMITTED FOR U.S. OCCUPATIONAL EXPOSURE (1981).

ACCORDING TO THE NATIONAL HOUSEHOLD PESTICIDE USAGE STUDY CONDUCTED BY EPA IN 1980, AN ESTIMATED 84% OF U.S. HOMES USE PESTICIDES INDOORS. BETWEEN 4% AND 5% OF THOSE HOMES ARE ESTIMATED TO HAVE OCCUPANTS WHO HAVE EXPERIENCED ACUTE TOXIC REACTIONS AFTER SUCH USE. IN COMMERCIAL PESTICIDE APPLICATIONS, THERE HAVE BEEN REPORTS OF ACCIDENTIAL INTRODUCTION OF THE CARCINOGENIC TERMITICIDE CHLORDANE INTO RESIDENCES WITH SUB-SLAB HEATING DUCTS, SOMETIMES GENERATING INDOOR CONCENTRATIONS SUFFICIENTLY HIGH TO INDUCE ACUTE ILLNESS, ACCORDING TO A GENERAL ACCOUNTING OFFICE STUDY IN 1980.

THE TREND TO BETTER INSULATED, "TIGHTER" BUILDINGS AND HOMES TO IMPROVE ENERGY EFFICIENCY HAS EXACERBATED THE PROBLEM CAUSED BY INDOOR AIR SOURCES. IT HAS BEEN FOUND THAT REDUCING VENTILATION IS A VERY EFFECTIVE MEANS OF REDUCING ENERGY COSTS. IN THE CASE OF HOMES, MOST VENTILATION OCCURS NATURALLY THROUGH OPEN WINDOWS. WHEN WINDOWS ARE CLOSED IN THE HEATING AND COOLING SEASONS, HOMES ARE VENTILATED ONLY BY INFILTRATION, OR UNINTENTIONAL LEAKAGE OF AIR THROUGH CRACKS AROUND WINDOWS AND DOORS AND LEAKS IN THE BUILDING FRAME, DRIVEN BY WIND-CREATED PARTIAL VACUUM AROUND THE HOUSE AND INDOOR/OUTDOOR TEMPERATURE DIFFERENCES. THIS ENABLES TOXIC POLLUTANTS IN THE AMBIENT AIR TO ENTER THE HOME AND REMAIN THERE FOR LONG PERIODS DUE TO INFREQUENT AIR EXCHANGES. AS DISCUSSED EARLIER, THE SAME APPLIES TO OFFICE BUILDINGS AND OTHER NON-INDUSTRIAL BUILDINGS WHERE ENERGY CONSERVATION EFFORTS HAVE LARGELY REDUCED AIR EXCHANGE RATES SIGNIFICANTLY.

THE INDOOR AIR ENVIRONMENT PRESENTS A UNIQUE MICROCOSM WHICH MIGHT BE CONSIDERABLY MORE COMPLEX THAN THE OUTDOOR ENVIRONMENT. ONE ASPECT OF THE DEBATE ON THE EXTENT TO WHICH INDOOR TOXICS POSE SIGNIFICANT HEALTH RISKS TO THE HUMAN POPULATION IS THAT MOST PEOPLE SPEND MORE TIME INSIDE A HOME OR BUILDING THAN THEY DO OUTSIDE IN THE AMBIENT AIR. ONE STUDY FOR THE ELECTRIC POWER RESEARCH INSTITUTE SUGGESTS THAT PEOPLE MAY SPEND AS MUCH AS 80-90% OF THEIR TIME INDOORS. ADDITIONALLY, CERTAIN POPULATION GROUPS SUCH AS INFANTS, CHILDREN, THE ELDERLY, AND THE INFIRM MAY BE MORE SUSCEPTIBLE TO ADVERSE HEALTH EFFECTS AND ALSO SPEND EVEN MORE TIME INDOORS.

RECALL THAT I HAD STATED EARLIER THAT THE INDOOR AIR PROGRAM WAS IN OF ITSELF NOT A REGULATORY PROGRAM. THE ORIGINAL GOALS STATED IN TESTIMONY I PRESENTED TO CONGRESS IN 1983 WERE:

- TO DEVELOP AN UNDERSTANDING OF THE MAGNITUDE OF THE RISK TO HUMAN HEALTH FROM EXPOSURES TO INDOOR AIR POLLUTANTS AND CONTRIBUTION OF VARIOUS ENERGY CONSERVATION MEASURES, INTRODUCTION OF NEW BUILDING MATERIALS AND CONSUMER PRODUCTS TO THIS RISK; AND

- TO PROVIDE TECHNICAL INFORMATION AND GUIDANCE ON INDOOR AIR QUALITY ISSUES, INCLUDING EFFECTIVE MITIGATION MEASURES, TO STATE AND LOCAL GOVERNMENTS, THE PRIVATE SECTOR, AND THE GENERAL PUBLIC.

CONSEQUENTLY, WE HAD INITIALLY SET EXPOSURE DATA AS A HIGH PRIORITY SO THAT WE COULD BETTER ESTIMATE RISKS. WE FELT WITH MORE SCIENTIFICALLY SOUND ALLOCATIONS OF RISKS BETWEEN A RANGE OF MICROENVIRONMENTS, AMBIENT, WORKPLACE OR INDOORS, WE WOULD BE BETTER EQUIPPED TO ESTABLISH RISK REDUCTION STRATEGIES, TO DETERMINE WHICH STATUTES WOULD BE APPLIED AND TO DETERMINE WHICH GOVERNMENTAL AGENCY (FEDERAL, STATE OR LOCAL) COULD DEAL WITH THESE RISKS MOST EFFECTIVELY. IN OTHER WORDS, WE WERE SUPPORTING THE RISK BASED REGULATORY APPROACH THAT CAME INTO BEING IN THE EARLY 80's.

THE NATIONAL ACADEMY OF SCIENCES ISSUED A REPORT IN THE EARLY 1980's THAT DESCRIBED THE RISK ASSESSMENT PROCESS AS BEING MADE UP OF FOUR INDEPENDENT STEPS: (1) HAZARD IDENTIFICATION; (2) DOSE-RESPONSE ASSESSMENT; (3) EXPOSURE ASSESSMENT; AND (4) RISK CHARACTERIZATION. I WOULD LIKE TO SAY A LITTLE ABOUT EACH STEP, AS THIS PROCESS HAS PROVEN TO BE A CONCEPTUAL FRAMEWORK FOR OUR RESEARCH PROGRAM.

CONCERNING "HAZARD IDENTIFICATION", THE HEALTH RESEARCH COMMUNITY HAS FOR MANY YEARS BEEN DEVELOPING A DATA BASE ON THE HEALTH EFFECTS OF POLLUTANTS. WE HAVE INFORMATION AVAILABLE OF THE HAZARDS OF SOME OF THE POLLUTANTS WE ENCOUNTER ON A DAY-TO-DAY BASIS BUT THAT DATA BASE IS LIMITED. FOR EXAMPLE, WE KNOW THAT ONLY 20 OR SO CHEMICALS HAVE BEEN IDENTIFIED AS HUMAN CARCINOGENS AND, AT THE OTHER END OF THE SPECTRUM, PROBABLY 2-3 DOZEN HAVE BEEN IDENTIFIED AS BEING NON-CARCINOGENS. THAT MEANS THAT WE KNOW VERY LITTLE ABOUT CARCINOGENIC PROPERTIES OF LITERALLY 1,000's OF CHEMICALS. MORE IMPORTANTLY THERE ARE OTHER HUMAN HEALTH ENDPOINTS WHICH WE KNOW EVEN LESS ABOUT: REPRODUCTIVE EFFECTS, NEUROTOXIC EFFECTS, TERATOGENICITY, MUTAGENICITY, AND A NUMBER OF OTHER HEALTH ENDPOINTS OF CONCERN.

THE SECOND STEP IS "DOSE-RESPONSE ASSESSMENT". WE KNOW SO LITTLE ABOUT THE DOSE-RESPONSE OF CHEMICALS IN HUMAN ORGANS AND TISSUE. FOR MANY OF THE CHEMICALS WE ENCOUNTER, WE RELY UPON ANIMALS AS SURROGATES. WHEN DOING SO, THERE IS UNCERTAINTY ASSOCIATED WITH EXTRAPOLATING FROM THE HIGH DOSES ADMINISTERED TO ANIMALS IN THE LABORATORY TO THE ENVIRONMENTAL CONCENTRATIONS GENERALLY EXPERIENCED BY THE HUMAN POPULATION. BUT NEVERTHELESS RISK ASSESSORS DO EXTRAPOLATE WITH THE BEST TOXICOLOGICAL DATA THAT CAN BE OBTAINED.

THE THIRD STEP OF THE RISK ASSESSMENT PROCESS IS THE EXPOSURE ASSESSMENT. WE THINK WE CAN MAKE SIGNIFICANT IMPROVEMENTS ON THE ACCURACY OF RISK ASSESSMENTS WITH AN IMPROVED EXPOSURE DATA BASE. EXPOSURE DATA IS EXTREMELY LIMITED. KNOWING THAT 80-90% OF OUR DAY IS SPENT INDOORS, RISK ASSESSORS BECOME CONCERNED THAT EXPOSURE ESTIMATES TO CERTAIN CHEMICALS MAY BE OFF BY ORDERS OF MAGNITUDE.

THE LAST STEP IN RISK ASSESSMENT IS RISK CHARACTERIZATION. THAT AMOUNTS TO PUTTING ALL OF THE DATA TOGETHER TO YIELD ESTIMATES OF DEATHS OR INCIDENCES OF A SPECIFIC EFFECT OCCURRING. THIS IS REFLECTED AS A NUMERICAL ESTIMATE WHERE DATA EXISTS, OR ON THE OTHER HAND A STATEMENT QUALIFYING RISKS IN A NARRATIVE FORMAT.

I BELIEVE THAT WHAT I HAVE BEEN SAYING POINTS OUT THE UNCERTAINTY ASSOCIATED WITH RISK CHARACTERIZATION. LACK OF EXPOSURE DATA, EXTRAPOLATION ERRORS AND HEALTH EFFECTS DATA ALL CONTRIBUTE TO THE UNCERTAINTY OF RISK ASSESSMENTS.

WE IN THE SCIENTIFIC COMMUNITY HAVE A RESPONSIBILITY TO STRIVE FOR ACCURACY AND MINIMAL UNCERTAINTY IN RISK ASSESSMENTS FOR A VERY IMPORTANT REASON RISK MANAGERS USE RISK ASSESSMENT INFORMATION IN CONJUNCTION WITH WHAT KNOWLEDGE IS AVAILABLE OF ENGINEERING, ECONOMICS, SOCIAL GOALS, ETC. TO DEVELOP REGULATORY STRATEGIES. THEY MIGHT BEGIN

TO ASK WHETHER OR NOT A DIFFERENT SET OF STRATEGIES THAN WE CURRENTLY IMPLEMENT MIGHT BE WARRANTED IF EXPOSURES OF RISKS ASSOCIATED WITH INDOOR ENVIRONMENTS ARE GREATER THAN THOSE EXPERIENCED OUTDOORS. THEY MIGHT BEGIN TO ASK WHICH STATUTE WOULD BE MOST EFFECTIVE TO REDUCE RISKS. THESE ARE ONLY HYPOTHETICAL QUESTIONS, BUT THEY ARE THE TYPE OF QUESTIONS THAT POLICY ANALYSIS MIGHT BEGIN TO ASK THEMSELVES ONCE WE, THE SCIENTIFIC COMMUNITY, PROVIDE THEM WITH THE ANSWERS ON TOTAL RISK AND EXPOSURES. WITHOUT THE KNOWLEDGE THAT WE CAN GAIN FROM RESEARCHING THE INDOOR AIR POLLUTION PROBLEM, WE CAN'T BEGIN TO DEVELOP ANSWERS TO ANY OF THOSE QUESTIONS OR MANY OTHERS THE POLICY ANALYSIS ADDRESS DAILY.

TO STUDY THE INDOOR AIR POLLUTION PROBLEM IN THE FEDERAL GOVERNMENT, SIXTEEN FEDERAL AGENCIES HAVE FORMED A COMMITTEE ON INDOOR AIR QUALITY RESEARCH AIMED AT IMPROVING THE COORDINATION BETWEEN THE VARIOUS AGENCIES. NOT ALL OF THESE AGENCIES ARE ENGAGED IN RESEARCH; SOME OF THEM ARE ONLY USERS OF THE INFORMATION. BUT, FOR THE MOST PART, EVERY ONE OF THEM IS VITALLY INTERESTED IN THIS ISSUE AND USES DATA IN ONE FASHION OR ANOTHER FOR THE CONDUCT OF ITS BUSINESS.

THE TASKS DEFINED BY THE INTERAGENCY COMMITTEE ADDRESS ALL FOUR FACETS OF THE RISK ASSESSMENT PROCESS: SOURCE CHARACTERIZATION, HEALTH EFFECTS STUDIES, EXPOSURE ASSESSMENT AND RISK ASSESSMENT. THE MAJORITY OF RESEARCH AT THE FEDERAL LEVEL IS PERFORMED BY THE DEPARTMENT OF ENERGY, THE TENNESSEE VALLEY AUTHORITY, THE BONNEVILLE POWER AUTHORITY, THE NATIONAL BUREAU OF STANDARDS, THE DEPARTMENT OF HEALTH AND HUMAN SERVICES, THE NATIONAL AERONAUTICS AND SPACE ADMINISTRATION, THE DEPARTMENT OF DEFENSE, THE CONSUMER PRODUCT SAFETY COMMISSION AND THE ENVIRONMENTAL PROTECTION AGENCY.

IT IS IMPORTANT TO NOTE THAT RESEARCH PERFORMED BY EACH ONE OF THESE AGENCIES IS IN ACCORDANCE WITH ITS OWN PARTICULAR MISSION. THEREFORE, YOU SEE DOE FOCUSING ON ISSUES RELATED TO ENERGY CONSERVATION, CPSC FOCUSING ON PRODUCT EVALUATION RELATED ISSUES, ETC. THE COOPERATION EXPERIENCED TO DATE HAS RESULTED IN INDIVIDUALLY AND JOINTLY FUNDED STUDIES BY FEDERAL AGENCIES TO ADDRESS SPECIFIC PROBLEMS.

PROGRESS HAS BEEN MADE. METHODS AND INSTRUMENTATION HAVE BEEN DEVELOPED WHICH ARE LESS INVASIVE THAN TECHNOLOGY USED IN THE PAST. WE NOW HAVE PASSIVE AS WELL AS ACTIVE MONITORS THAT CAN BE PLACED IN HOMES OR BE CARRIED BY PEOPLE TO OBTAIN A MORE ACCURATE MEASUREMENT OF THE DOSES RECEIVED BY HUMANS. CONSEQUENTLY, WE CAN BETTER ESTIMATE THE TOTAL EXPOSURE.

AN IMPORTANT AREA OF INTERAGENCY COOPERATION CONCERNS RADON. RADON RISKS CAN BE MITIGATED. EPA HAS DEMONSTRATED THAT, DEPENDING ON THE BASIC DESIGN AND CONSTRUCTION PRACTICES OF HOMES IN THE READING PRONG AREA, TECHNOLOGY CAN REDUCE ELEVATED RADON EXPOSURES TO LESS THAN 0.02 WL OR 4 PICOCURIES/LITER. CURRENTLY, EPA IS DISCUSSING A JOINT PROJECT WITH BONNEVILLE POWER AUTHORITY (BPA) TO DEMONSTRATE MITIGATION TECHNOLOGY IN THEIR SERVICE AREA. SEVERAL STATES AND COMMUNITIES HAVE

REQUESTED HELP FROM EPA's OFFICE OF RESEARCH AND DEVELOPMENT TO DIAGNOSE THE MAGNITUDE OF THEIR SPECIFIC RADON PROBLEM AS WELL AS DEMONSTRATING MITIGATION TECHNOLOGY. INCIDENTIALLY, AND VERY IMPORTANTLY, THE GOAL OF THE EPA's R&D STAFF IS TO DEMONSTRATE THAT MITIGATION IS POSSIBLE AT A COST THAT CAN BE AFFORDED BY MOST HOMEOWNERS. WE NEED MORE LONG TERM ASSESSMENTS TO SEE IF WE ARE ACHIEVING OUR GOAL.

IN A MORE GENERAL NOTE, MODELING STUDIES BY NBS AND DOE SEEM TO BE PROVIDING A DIAGNOSTIC CAPABILITY TO LINK MANY PARAMETERS TO PRODUCE PREDICTIONS OF INDOOR AIR QUALITY AS A FUNCTION OF EXCHANGE RATE, SOURCES, ETC. A LARGE DATA BASE IS NEEDED TO VALIDATE THESE TOOLS.

IN ANOTHER ARENA, TVA, HHS, CPSC, EPA AND OTHERS ARE CONTINUING TO WORK TOGETHER TO EVALUATE THE EFFECTS OF ELEVATED CONCENTRATIONS OF POLLUTANTS FOUND INDOORS ON HUMAN HEALTH BY JOINTLY FUNDING TARGETED HEALTH STUDIES. EPA IS EVALUATING BIOLOGICAL MARKERS TO DETERMINE IF BETTER MEASURES OF DOSE OR BODY BURDEN CAN BE OBTAINED. EPA HAS PREPARED DESIGNS FOR A MULTIPOLLUTANT EXPOSURE SURVEY; HOWEVER, PLANS FOR THE SURVEY ARE NOW ON HOLD.

CONGRESSIONAL INTEREST HAD BEEN STRONGLY ORIENTED TOWARD THE CONDUCT OF A MULTIPOLLUTANT EXPOSURE SURVEY. THE ORIGINAL PEER REVIEW OF EPA's RESEARCH PROGRAM STATED IN NO UNCERTAIN TERMS THAT THE "SURVEY" SHOULD BE THE FOCUS OF EPA's PROGRAM. THE 1981 NAS REPORT ON INDOOR AIR ALSO SPOKE OUT IN FAVOR OF A NATIONAL SURVEY TO ASSESS THE MAGNITUDE OF THE PROBLEM.

CURRENT FEELING IN CONGRESS, THE SCIENTIFIC COMMUNITY AND SOME FEDERAL AGENCIES THAT CONDUCTING A NATIONAL MULTIPOLLUTANT SURVEY MIGHT NOT BE PRUDENT AT THIS TIME. THERE IS A GROWING CONCERN THAT A MORE STRATEGIC APPROACH IS NEEDED TO PRIORITIZE THE POTENTIAL RISKS OF POLLUTANTS FOUND INDOORS. CONSEQUENTLY, WE ARE TAKING A STEP BACK TO PUT INTO PLACE A SERIES OF TASKS THAT WILL LEAD TO A SHARPER FOCUS OF OUR RESEARCH PROGRAM. PRIOR TO ANY FINAL DECISION REGARDING FUNDING A LARGER SCALE SURVEY, THE OFFICE INTENDS TO PROCEED WITH A PHASED APPROACH, I.E., CONDUCT A SCREENING SURVEY TO ASSESS THE RANGE (DIVERSITY) OF POLLUTANTS FOUND AND THEIR RELATIVE MAGNITUDE (SORT OF AN EXPANDED PILOT SURVEY) OF A NUMBER OF HOMES THAT WOULD NOT NECESSARILY REPRESENT A STATISTICALLY VALID SAMPLE SIZE. IN THE MEANTIME WE WILL PREPARE AN ASSESSMENT OF THE EFFECTS AND SOURCES OF POLLUTANTS BASED ON EXISTING DATA BASES OF INDOOR POLLUTANTS. AFTER CONDUCTING THE SCREENING SURVEY AND DEPENDING ON THE "TIGHTNESS" OF THE DATA AND CHEMICALS FOUND, WE WOULD USE THE SURVEY/ASSESSMENT PHASED APPROACH TO GUIDE DECISION MAKING ON WHETHER TO PROCEED WITH ADDITIONAL SMALL-SCALE SURVEYS OR TO DIRECT ADDED ATTENTION TO SOURCE WORK AND/OR TARGETED HEALTH STUDIES OR FUND A LARGE-SCALE FIELD SURVEY. MY CONCERN IS THAT OUR RESEARCH APPROACH BE SCIENTIFICALLY SOUND AND POLICY RELEVANT. CONSEQUENTLY, THIS PLAN WILL BE SUBJECTED TO PEER REVIEW.

NEEDLESS TO SAY, I HAVE FOUND THE INDOOR AIR ISSUE EXTREMELY DYNAMIC. THE PERSONALITIES OF INDIVIDUALS SEEM TO BE AS MUCH A PART OF THE ISSUE AS THE SCIENCE OR INTEREST THEY BRING TO THE ISSUE. CERTAINLY, THE ISSUE WILL NOT BE FORGOTTEN. I BELIEVE WE WILL SEE MAJOR ADVANCES IN OUR ABILITY TO FACTOR INDOOR AIR EXPOSURES INTO TOTAL RISKS. I ALSO EXPECT THAT WE SHALL SEE MORE SUCCESS STORIES SUCH AS RADON RISK MITIGATION.

THIS CONFERENCE PRESENTS APPROXIMATELY 16 SESSIONS DEDICATED TO ALL FACETS OF THE INDOOR AIR ISSUE. THERE ARE ALSO PARTICULAR SESSIONS, SUCH AS THAT DEVOTED TO RISK ASSESSMENT, ETC., THAT BEAR UPON THIS ISSUE. MY COMPLIMENTS TO APCA FOR SPOTLIGHTING INDOOR AIR IN 1986. WITHIN THE NEXT 2-3 YEARS, I HOPE TO SEE MORE SESSIONS DEDICATED TO INDOOR AIR, HOW TO MORE ACCURATELY ASSESS ITS RISKS AND MOST IMPORTANTLY, WHAT TO DO ABOUT THEM.

WE MUST LEARN HOW TO COMMUNICATE WHAT WE LEARN ABOUT RISKS AND RISK MITIGATION. COMMUNICATION OF RISKS IS A VITAL ELEMENT, BUT PERHAPS THIS TOPIC SHOULD BE LEFT FOR ANOTHER TIME. FOR NOW I CAN ASSURE YOU THAT MY OFFICE WILL CONTINUE TO COOPERATE WITH APCA TO BRING RESULTS OF FEDERAL RESEARCH TO THE ATTENTION OF THIS GROUP SO THAT WE CAN WORK OUT STRATEGIES TO REDUCE RISKS.

THE END

HARVARD UNIVERSITY
SCHOOL OF PUBLIC HEALTH

DEPARTMENT OF ENVIRONMENTAL SCIENCE AND PHYSIOLOGY
PHYSICAL SCIENCES AND ENGINEERING PROGRAM

665 HUNTINGTON AVENUE
BOSTON, MA 02115

January 20, 1984

Dr. Donald J. Ehreth
Deputy Assistant Administrator
Office of Research and Development
U.S. EPA
401 M Street, SW
Washington, DC 27711

Dear Dr. Ehreth:

We are pleased to submit the finding of our Ad Hoc Review Panel "An Agenda for Research: Indoor Environments." The task was difficult. Quantifying the health and comfort-risks associated with indoor environments and their contribution to total human exposures is presently constrained by current funding levels. The Agency is commended for its recognition of the integral role that exposure assessment plays in risk management. Our contribution is in direct response to William Ruckelhaus'address before the National Academy of Sciences ("Science, Risk, and Public Policy", June 22,1983), in which he requested involvement of the scientific community in the management of environmental risks. Knowledge of contaminants in our indoor environments, their sources, behavior, concentrations and control is fundamental to the process of overall risk management. We must demonstrate the effectiveness of our standards and control strategies on mitigating human health risks. No longer can we afford to nearsightedly examine only a fraction of our population exposures. Therefore, the Agency is urged to conduct an indoor air quality research program that includes:

- A national survey to characterize the nature and distribution of harmful contaminants encountered, in part, in the residential environment. Initially, this survey would include carbon monoxide, nitrogen dioxide, size fractionated particulates, chlorinated hydrocarbons, formaldehyde and radon.
- Source characterization studies should include indoor combustion sources, pesticides, organic compounds from building materials and consumer products, and radon from water, soils, and building materials.

With respect to the contaminants in bullet ①?

- Detailed studies should be conducted that determine the behavior and interactions among contaminants and the indoor environment.

- Measurement materials, both hardware and questionnaires, are needed to help the current data bases.

- The Agency is encouraged to assist both intra- and extra-mural research related to health assessment on indoor contaminants. Many of these research programs are justified under base-program funding, therefore, the Indoor Air Qualit Research (IAQR) effort should be limited initially to "add-o that refine the usefulness to an integrated program on indoo environments.

Good

Speaking on behalf of the panel members, we found this review process stimulating. The willingness of EPA to challenge the panel with multiple options encouraged us to believe that our contribution would be timely and vital to the formulation of the Agency's research direction.

Sincerely,

John D. Spengler

John D. Spengler [illegible]
Associate Profess~~or in~~
Environmental Health Science

An Agenda for Research:
INDOOR ENVIRONMENTS

Report of the Ad Hoc Review Panel
on
U.S. Environmental Protection Agency's
Indoor Air Quality Research Program

January 20, 1984

Summary of Findings and Recommendations
of
the Review Panel Meeting at the
Energy and Environmental Policy Center
Kennedy School of Government
Harvard University
December 8 and 9, 1983

FINAL REPORT JANUARY 20, 1984

An Agenda for Research:

INDOOR ENVIRONMENTS AD HOC REVIEW PANEL ON US EPA'S INDOOR AIR QUALITY RESEARCH PROGRAM

Objectives:

1. To assemble experts from diverse backgrounds to review EPA program plans for indoor air quality research in FY '84 and F '85.

2. To report on the scientific and technical merit of the overall indoor air quality research (IAQR) plans as well as o specific program elements.

Under the auspices of the Energy and Environmental Policy Center, Kennedy School of Government, Harvard University, a group of 40 scientists, engineers, and health researchers and other interested individuals assembled at Harvard's Kennedy School of Government on December 8 and 9. The purpose of the meeting was to review the US EPA's research program on indoor air quality. On Thursday, December 8, EPA presented three research programs that its offices had developed in response to a Congressional mandate and appropriation. Dr. Don Ehreth charged the review panel with an exploration of two overriding questions regarding the adequacy of the plans: 1) Will the plans for indoor air quality research enhance EPA's ability to characterize health risks? 2) Will they provide a better understanding of the nature of the various indoor air quality problems across the nation so that the private sector and state and local governments can more objectively take action to protect the public when mitigation measures are needed and feasible.

Following formal presentations, the group divided into subgroups to address specific elements and issues of EPA's indoor air quality research program. The sub-committees met on Thursday, December 8 and Friday, December 9. Verbal presentations of subcommittee reports were made in a plenary session on Friday. Following the review meeting, co-chairmen and individuals prepared written reports and comments included in this report. The draft and final report were reviewed by several members (approximately 25) of the review group.

The consensus has been reached that a concerted, well-funded, and integrated multi-year indoor air quality research program of national scope should be undertaken. However, there is a need for immediate near-term results targeted at contaminants with high indoor concentrations affecting human exposures and having either; 1) potentially adverse health effects, or; 2) important implications for air quality standards and emission controls of pollutants regulated by EPA. These near-term objectives must be balanced with appropriate attention to long-term needs for methods development, studies on pollutant behavior and interactions in indoor environments, clinical laboratory exposure chamber and human epidemiologic health studies, as well as investigation of pollutant control strategies and the policy issues.

Indoor environments present a challenging opportunity for research. The chemical, physical, and biological airborne contaminants found indoors are far more diverse than those found outdoors. The number and types of indoor environments occupied or frequented by people are equally diverse. We have only just begun to quantify the relative contributions to total human exposures that can occur from indoor sources. The evidence to date strongly suggests that the indoor components are important and, at times, dominate all other exposure components.

Management of environmental risk in an equitable and effective manner demands a substantial indoor air quality research effort supported by the federal government. To date, no systematic or sustained indoor air quality research program exists within the federal government. It is true that the Department of Energy, Consumer Product Safety Commission, Department of Defense, HUD, TVA, NIOSH, EPA and others, have sponsored research programs related to indoor environments and human exposures. However, a comprehensive program involving multiple pollutants, their sources, dynamic behavior, field characterization and health consequences and control strategies has not been explicitly undertaken as an agency research program. The scientific community appreciates the indoor air quality research program now initiated by EPA. We have high expectations that if it is integrated with public and privately funded research programs, and if it is sustained with increased support over a ten-year period, we will have the information needed to:

1. Provide incentives to stimulate mitigation measures in the private sector.
2. Provide state and local governments with information and resources such as design data or monitoring, to help redress

existing building problems and insure acceptable indoor air quality in future buildings.

3. Provide Congress and regulatory agencies with cost-effective environmental management programs that protect the public fr harmful exposures.

It is the intent of this report not to criticize the plans presented, but to offer constructive guidance to the agency on how it might best achieve both near-term and long-term research objectives. This report offers concise recommendations on EPA's indoor air quality research plans at two levels: 1) Overview on the general direction and management. 2) Specific recommendations by program elements.

GENERAL OVERVIEW:

1. The data base characterizing the distribution of the gases, vapors and particles in the built environment is currently insufficient for an adequate evaluation of the potential health risks. Evidence to date suggests that segments of our population are being exposed to elevated pollutant concentrations inside their homes, their offices, and other types of buildings. Therefore, a national multi-pollutant study to broadly characterize the extent of these exposures is needed.

Mindful of the varying nature of source use, source distribution, ventilation rates, occupant and structural factors, sampling should be integrated across days or weeks. (Time-resolved information is required as well in order to correlate highly variable indoor levels with specific activities and sources. See other recommendations.) Vent[illegible] ables should be incorporated into a survey. Each indoor contaminant considered should be justified on the basis of its potential health effect and its relevance to total human exposures. The contaminants investigated should be pertinent to the pollutants currently under the mandate of the agency through its enabling authorities. The panel believes that the contaminants meeting these criteria and deserving careful consideration are radon and radon progeny, formaldehyde, halogenated hydrocarbons (including chlorinated and brominated compounds) and the criteria pollutants: carbon monoxide, nitrogen dioxide and suspended particulate matter less the 10 micrometers in aerodynamic diameter (PM^{10}) including a measure of the fine fraction. Certainly, there are other indoor contaminants that are of concern:

asbestos fibers, PNA's, nitrosamines, and organophosphates, among others. However, mindful of what is feasible and urgently needed from a broad-based survey, the list was limited to the contaminants cited.

2. In recent years, EPA has sponsored extensive studies on human exposures to carbon monoxide and volatile organic compounds (VOC). The review panel recommends that these studies soon be thoroughly evaluated. The TEAM VOC projects and the Washington and Denver carbon monoxide exposure studies may offer an opportunity to understand how pollutant concentrations vary: 1) within several indoor environments and 2) across source structures, and 3) how human factors influence those concentrations over a short term period. We underscore the need to fully evaluate existing data; the panel believes that these and other indoor studies might be useful in the statistical design of future single- and multi-pollutant characterization studies.

3. The development of health effects information is expensive and cannot be substantially addressed under current funding levels. Further, it should be anticipated that fruitful epidemiologic investigations may involve more than two years of research activities. Therefore, it is prudent to consider activities beyond two years, and to identify existing opportunities within other agencies. Several such opportunities exist through research programs currently sponsored by the National Institute of Health (Environmental Health Sciences, and the Heart, Lung and Blood Institutes), US EPA (HERL), Electric Power Research Institute, Health Effects Institute, Department of Energy and perhaps other programs. EPA is encouraged to assess these ongoing

studies to determine if they are pertinent to EPA's IAQR needs. Where needed, EPA should support additional measurements or analyses in existing clinical health studies and epidemiologic surveys. The health significance of indoor exposures to passive smoking, nitrogen dioxide, carbon monoxide, and radon might be advanced through EPA's involvement within investigations that are currently underway.

Other survey programs should be explored to obtain additional information on health effects of indoor pollutants. The National Center for Health Statistics (NCHS) conducts both the National Health Interview Survey (NHIS) and the National Health and Nutritional Evaluation Survey (NHANES). Serious planning efforts could begin now between EPA and NCHS for expanding these surveys to include indoor air quality information. For example, NHANES housing characterization survey questionnaire could be modified to include better descriptor variables of potential indoor air quality pollution sources or factors. The planned multi-pollutant IAQ study could be coordinated such that it might provide better characterization of the targeted population for the 1987 NHANES survey. For instance, the NHANES survey could include specific questions on health problems related to indoor pollutants.

4. Currently, indoor air quality is not the explicit focus of a great deal of sponsored research. As such, it is difficult for an individual to be aware of all of his/her organization's activity. Often, those outside the agency have an advantage through formal and informal exchanges to obtain a broader perspective across program areas both within the public and private sectors. It is therefore recommended that the interagency task force on

indoor air quality be expanded to include 5 to 10 outside advisors from academic research organizations, national labs, health or environmental foundations, private industry (if appropriate), as well as manufacturing research groups.

5. In spite of a modest federal research program on indoor air quality, a surprising number of investigations are conducted or being planned. EPA, other federal agencies, states, industrial associations, foundations, and other organizations, are currently engaged in research which relates to indoor environments. It is essential that EPA IAQR program staff and management remain abreast of these activities so that, with limited funds, EPA may target and adapt its research efforts while monitoring these extra-mural research activities. EPA's IAQR staff should be encouraged to participate on research review and advisory panels as a mechanism to interact with other ongoing projects. EPA can also play a leadership role in sponsoring workshops that directly foster scientific and technical exchange among these programs. A permanent advisory committee and peer review program should be devised to evaluate and integrate the results of both intramural and extra-mural indoor air quality research, and to evaluate developing research plans. In two-years' time, EPA should consider commissioning another assessment study on indoor air quality that will update the 1981 National Academy of Science report.

6. EPA's IAQR program makes an attempt to address all the important aspects of indoor air quality with a few noted exceptions. A broad-based, completely comprehensive program is not justified at present. First, other existing or planned research

programs, both within the agency and outside, have not been assessed to determine if they might meet EPA's needs with little or no modification through some additional EPA funding. Second, a number of the projects described in these plans really need the commitment of multi-year funding to insure continuity and to prevent fragmentation. While we are encouraged by some degree of intra-agency integration, it is not clear from the plans presented whether indoor air quality research dollars will be spent offsetting program dollars in EPA's existing or planned research. Both the bioassay testing of emission source particulate extracts and some of the radon projects appear as examples of research that should be part of EPA base programs funded in FY '84 and FY '85. Hence, the IAQR program should not be utilized as additional sources of funds for these programs unless it can be convincingly demonstrated that they are an integral component of the required data base needs.

PROGRAM ELEMENTS:

A. Methods Committee - Co-chairs, Dr. David Fine and John Girman

The panel on methods adopted a broad definition to include method development, instrumentation, sampling survey design, and modeling. Relevant contributions from other groups or participants have augmented the recommendations presented under this and other sections. Because of the time limitations, this group did not discuss methods or instrument needs related to radon. Absence of specific recommendations does not constitute acceptance or rejection of EPA's IAQR program for radon instrumentation by the committee.

EPA should concentrate on establishing the necessary foundations for supporting future intercomparable studies by insuring that reliable instrumentation, operating procedures, and analyses are available. The devices that now exist and those that will be developed should be tested in a series of controlled laboratory and field condition experiments. These would include bench top, side-by-side comparisons in laboratories, and comparisons in chambers where conditions can be controlled. We suggest further verification by comparisons in live-in-laboratories (model homes) where conditions may vary but can be monitored. Such model home studies provide the possibility of exploring interferences where there are multiple sources and multiple interferents.

1. We recommend improvement in both active and passive monitoring for the following contaminants: formaldehyde, nitrogen dioxide, water vapor, polynuclear aromatic compounds, volatile organic compounds, and particulate matter (PM^{10}), including a

measure of the time fraction (< 2.5 micrometers).

2. EPA should initiate multi-pollutant pilot studies using model homes as the test facilities for the comparison of both active and passive sampling equipment. These model homes have the advantage of actually simulating real-life behavior patterns that incorporate multiple source emissions which may have multiple removal pathways. Model home studies where sources and their use patterns are carefully monitored would be helpful in identifying interferences that might be missed in laboratory settings.

3. EPA should validate both active and passive collection devices for a whole range of volatile organic compounds. There is also a particular need for collection devices to sample the less volatile, high molecular weight organic compounds. In general, there are concerns about the cost-effectiveness of using Tenax or any other collective medium in a variety of indoor locations for collection and identification of a spectrum of organic compounds. Cost-effectiveness must be evaluated in comparison to other techniques that utilize portable analytical equipment and/or identify a class of compounds, such as PNA, in a screening fashion for later follow-up to identify. The phased assessment programs developed for industrial source sampling may serve as an effective model.

4. There is a tendency to make QA/QC functions a controlling issue. In the case of indoor air pollution research, careful balance must be struck. There are uncertainties in health effects and great variability in indoor concentrations. The cost and adequacy of quality assurance and quality control must be balanced against the study design and purpose. For instance,

twenty percent accuracy on some passive monitors may be a cost-effective and adequate way of documenting the concentrations in a large number of homes. In any event, careful attention must be paid to systematic biases that might result from interferences, sample location selection, sample instabilities, laboratory techniques and sample selection techniques, among others. EPA should develop or support consensus efforts among the research community to standardize sampling protocols, define QA/QC requirements, design environmental survey questionnaires and indoor air quality health questionnaires.

5. One of the most immediate priorities is the development of a PM^{10} sampler compatible with indoor-residential applications. The presently available dichotomous samplers are cumbersome and require isolation of the pump because of high noise levels. The NBS personal dichotomous sampler is not suitable as a reliable, rugged, particulate monitor. Utilizing advances in microcircuitry, PM^{10} monitors as well as other monitors should be combined with other real-time sensors to provide continuous data as well as time-integrated data. The PM^{10} sampler should be designed to provide adequate samples for both mass and composition analyses.

6. Simulation models are viewed as important tools. The model development process tests our understanding of the physical environment and expands the analytical framework of our understanding. Properly validated models are essential for extending the results of research beyond the simple characterization of the sample population. At this time, the available indoor air quality models are reasonably well-suited only to describe the mixing of

single pollutants in a single compartment. Current models have limited ability to treat multiple, interacting pollutants, such as ozone and nitric oxide, in realistic physical situations. Improvements will depend on better characterization of mixing and air flow within the home, quantification of pollutant sinks and the reactive chemistry that occurs in the air and on surfaces. Combined energy use (conservation and thermal comfort), air infiltration, and indoor air quality simulation models are needed. These models could be provided to the architectural and construction professionals and be utilized by the Building Construction Standards Boards in the design of energy efficient structures, thus allowing for voluntary control of indoor air quality.

Plan A designated $250,000 for modeling while Plan B does not specifically designate funds for model development. We recommend that on the order of $100,000 be designated in support of multi-pollutant model development. It should be noted that several organizations are currently engaged in indoor air quality modeling developments. The incorporation of these groups into either a technical subcommittee on modeling or their participation in sponsored workshops on modeling would be a vital contribution to integrating the activities currently underway with EPA's indoor air quality research programs.

B. Field Monitoring Committee - Co-chairmen Drs. Jack O'Neil and David Grimsrud

The committee on monitoring dealt almost exclusively with the design aspects of an indoor characterization study of residences. It was felt that the IAQR FY '84 and FY '85 appropriations should be used to provide substantial support to coordinate a national program of high visibility and high information payback by characterizing indoor concentrations in a large number of residences across the U.S. Such a national survey is difficult to support under funding from other agencies or non-governmental organizations. It was noted that several small studies of indoor pollutant concentrations have been concluded or are in progress. The results of these previous studies must be reviewed prior to the final design of a national survey to characterize the distributions of pollutant concentrations found in the U.S. housing stock.

1. Two field monitoring designs are proposed in Plans A and B. While each had its own internal projects that represent careful design considerations, we find strong support for the concept of large, multi-pollutant sampling design as the initial step in the national survey. The national survey should have an explicit set of three steps of phases in its initial design. These are: a large, multi-pollutant screening survey for pollutants, perhaps using passive instrumentation; a follow-up study on a fraction of the homes that were sampled initially in which significant concentrations of the target pollutant were observed; and an additional study to determine the efficiency of mitigation efforts. It is recommended that passive integrating samplers be used to minimize costs, provided they have been adequately tested prior to the survey period.

The national screening survey should include radon (where geologic mapping indicates significant sources), carbon monoxide, and nitrogen dioxide (to represent unvented indoor combustion processes), formaldehyde and halogenated compounds. In these studies ventilation should be monitored using a perfluorocarbon tracer system. With no replication of these devices at a test site, a sampling package might be available for approximately $100, including analyses. Other pollutants should also be studied. However, until adequate, inexpensive samplers are developed, additional techniques could cause an excessive burden on the sampling design and would result in a significant reduction in the number of sites that can be screened.

The national survey sampling design should be flexible. Regional variations in sources and building factors should be taken into account. Some contaminants may only be monitored on a regional basis or on less than the full number of homes in the entire survey. A subset of homes would be monitored more frequently throughout the year to determine seasonal behavior patterns in the indoor concentrations. A smaller subset of homes should be sampled using area monitors to determine spatial variation of selected pollutants related to activities (especially cooking).

If inexpensive sampling packages are available, groups of trained volunteers could assist in deploying and retrieving samples. University science teachers, college students, utility auditors and community health personnel are all potential trained volunteers who could implement a master protocol on a regional basis.

Given these strategies and objectives, on the order of 5,000 houses could be monitored within a budget of $1 to 1.5 million over the next two years. Most of these homes would be sampled only once with subsets being monitored more frequently. At least two seasons need to be investigated -- summer and winter, that is, about 2,500 homes in each season. Assuming five geographic regions, then 500 homes would be considered each season in each region. Monthly average values for the primary measurements would provide a useful time scale for intercomparing results from all regions and seasons. Assuming four winter months (December to March) and four summer months (June to September), 125 homes per month in each region would be tested. Perhaps 5 to 10 homes would be selected for replicate measurements in subsequent monthly periods to determine seasonal representativeness. At least some of these homes should be selected on the basis of their detailed study in other ongoing IAQR programs for the purpose of quality assurance.

2. The second phase of the national survey should contain provisions for follow-up inspections and possible additional monitoring, both integrated and continuous. Initially, the target group for this follow-up study would be those homes which showed excessive pollutant concentrations in the initial survey. The need for additional monitoring beyond the portable lightweight screening equipment would be determined following inspection of sources and by interviewing occupants. We suggest that additional integrated monitoring might be more instructive than expensive continuous monitoring.

3. Results of a national indoor air quality characterization study could provide candidate homes where mitigation strategies

could be tested. On a case-controlled bases, the effectiveness of providing abatement information to the resident, real time sensors, air-to-air heat exchange, exhaust vent use, filters, absorbers, or other controls could be tested on a comparative basis.

4. We recognize a concern about organic chemicals in indoor spaces. Documenting the concentrations that result from the use and misuse of pesticides, insecticides, herbicides (primarily halogenated compounds) in restaurants, offices, and residences should be an objective of a detailed survey. However, for most volatile organic compounds there is tremendous variation of concentrations both in space and time. In part, this has already been demonstrated in previous studies such as TEAM. We recommend an intensive analysis of a limited number (about 25) of residential or commercial spaces identified by questionnaire as high-use facilities for pesticides and other consumer volatile organic containing products. This effort should identify the kinds and the strengths of sources in the same residential environments. Such a study might contribute to our understanding of the relative importance of indoor exposures if it is conducted and coordinated as part of a demonstration project which samples in the vicinity of known hazardous organic waste emitters. Given the public's concern for potential exposures around hazardous waste sites, a study of indoor and personal exposures to a variety of volatile organic compounds is urgently needed. EPA is well-advised to use a portion of the IAQR appropriations as seed money to start such projects.

C. LABORATORY/CHAMBER STUDIES COMMITTEE - Co-chairs Drs. Brian Leaderer and Demetrios Moschandreas

This subcommittee considered controlled studies of sources and pollutant behavior conducted in laboratory chambers or in model homes and buildings. Again, it was underscored that considerable work is already in progress and it is supported by other federal agencies as well as non-governmental institutions. We recommend that information from prior tests and these ongoing studies be continuously integrated into EPA's program. Early on, EPA should sponsor a workshop dealing specifically with source characterization as a mechanism for integrating EPA's efforts into other studies. We support EPA's plans for the study of control technologies. Since much of the control technology research funded under other programs is directed at single pollutants [illegible] it is felt that EPA does have a role [illegible] on indoor contaminants controls. We concur with EPA's primary focus on source testing with a secondary emphasis on pollutant behavior and removal.

The construction of a separate experimental test facility at EPA for conducting research on source emission rates is not justified, given the tentative nature of two years of funding and the unnecessary duplication of existing programs. Once an adequate baseline support of indoor air quality programs is obtained and guaranteed within EPA, the building of an in-house facility at RTP might be reconsidered. Since Plan B described the construction of a test facility as part of its programs on source testing, considerable savings could be achieved by eliminating this item. While we felt that the Plan A's $300,000 for source characterization over two years might be too low, the $800,000 recommended by Plan B is considered too high.

Source characterizations and studies of pollutant behavior could start almost immediately if EPA utilizes existing facilities. A modest $400,000 effort over two years should result in useful information that would integrate into ongoing field studies.

2. We recommend a number of contaminants be investigated in source characterization studies:

1. Since volatile organic compounds are very inadequately understood, controlled laboratory test studies need to be conducted as well as actual "live-in" home studies. At present there is insufficient information available to prioritize a list of specific volatile organic compounds (VOC's) or even classes of VOC's . Certainly concern based upon known health effects exists for classes of compounds such as pesticides, solvents from building materials and consumer products, and by-products produced from combustion. However, only limited information exists regarding the duration, frequency and magnitude of exposure to these classes of compounds. Early studies should focus on these uncertainties. Thus, while the review panel cannot provide detailed information about specific classes of VOC's that should be studied, the panel can suggest an approach that will provide this information.

A review of EPA's TEAM Study can suggest some of the VOC's which warrant further study. However, it must be recognized that the sampling method employed in this study, Tenax, is not satisfactory for all VOC's and the data from the TEAM Study is deficient with respect to both highly volatile organic compounds in the solvent range and the less volatile compounds of higher molecular weight. Emphasis should be given to studies which address these compounds to close an information gap.

Studies of organic emissions from building materials should be reviewed to determine which VOC's occur frequently and should receive further study. It is important to distinguish those VOCs which are of "bioeffluent" origin versus anthropogenic materials such as pesticides, solvents, etc. The so-called "bioeffluents" of people span many classes of VCC, namely, aromatics, ketones, acids, aldehydes, esters, and alcohols. These clearly must be distinguished from household components and VOC's from combustion sources.

Product information can be obtained from the CPSC regarding frequency of use, the prevalence of use, and the chemical composition of products to assist in establishing priorities with respect to chemical classes. Finally, the information obtained from the steps outlined above must be assessed in the context of the health effects associated with the chemical classes of compounds in assigning priority for study. Health effects information should be obtained from EPA's own staff, CPSC, NIH, and NIOSH.

2. Inorganic and organic speciation of particles emitted from combustion products should be further characterized for a variety of sources. This will allow source identification

using source-receptor techniques.

3. EPA needs to evaluate inorganic compounds and gases in combustion products from a variety of sources as generated under different operating modes. It will be important to field verify the emission rates determined from combustion appliances tested under laboratory conditions. Among the contaminants of concern are nitric oxide, nitrogen dioxide, and carbon monoxide. There is evidence that nitric oxide (NO) behaves differently in the indoor environment and may have health consequences.

4. Radon and radon progeny emissions from building materials, from soils, and water need to be studied further, a water emission factor needs to be determined.

5. Formaldehyde emissions from furnishings and other building products and the interaction with the indoor environments (i.e., source-loss mechanisms) should be further characterized in coordination with considerable work which has already been done in this area. Radical changes can occur over a one-to-five year period in emissions from resins.

6. Pesticides should be studied to understand the influence that their volatility has on indoor concentrations and the dynamics of their emission rates. Chlorinated hydrocarbon and organophosphate pesticides could be sampled as a class, possibly by selecting an adsorbent and analysis scheme which would separate the pesticides from common, innocuous freons.

ogy of indoor pollutants, it will be necessary to rely on questionnaires for characterizing the health responses and environmental conditions related to indoor exposures. These questionnaires should be short and simple. They could be used in conjunction with national monitoring studies, health interviewing surveys, and environmental inventories. In general, tested and accepted health and environmental questionnaires will enhance their joint use in future surveys.

4. The National Academy of Sciences report on Indoor Pollutants (1981) and the WHO/EURO report on Indoor Pollutants: Exposure and Health Effects (1983) identified passive tobacco smoke exposure and combustion emissions, among other pollutants, that warranted further health studies. Specifically, indoor concentrations of particles, nitrogen oxides and carbon monoxide need to be studied further. EPA can help evaluate the tobacco smoke component of indoor aerosols, as well as other particles, nitrogen oxides and carbon monoxide, in various other studies. More specifically, EPA's IAQR program can assist the efforts of NIEHS, NCHS, NHLBI by improving instrumentation, conducting exposure studies, and in general, providing quantitative information about particle size and composition in the indoor environments, so that ongoing studies (supporting by these other sources) could better evaluate exposure - health response relationships.

5. The plans do not give any evidence that there has been coordination, or any integration with research activities of other organizations, such as NIEHS, NHLBI,

NIOSH/CDC, CPSC, DOE, GRI and EPRI. In particular, EPA is participating in health effects research through co-sponsorship of the Health Effects Institute (HEI). HEI is supporting or considering research projects that could provide some information on the health effects of indoor pollutants (CO and NO_2) that are also emitted by mobile sources. EPA may enhance the information relevant to indoor environments through additional support to HEI, NIEHS, NHLBI, EPRI or other health studies.

The organization of IAQR plans in a framework that systematically leads to health risk and policy assessments must be considered from the outset. As shown in Figure 1, EPA's IAQR plans are not conceptualized in a logical and integrated framework. The plans presented appear fragmented and deal primarily w[illegible] and field studies. We argue that some investment be made in quantitative risk assessment to help program leaders to continually evaluate and integrate the results of federally-funded research. Establishing this specific function early-on should facilitate coordination of intra- and extra-mural IAQ programs.

6. The scientific leadership of the IAQR program is critical. The panel is distressed by the internal struggles to direct and manage the EPA IAQR program. By its very nature, indoor air quality research cuts across many program areas and many disciplines in the Agency. The scientific leadership of the program must be familiar with related research within the Agency and related research supported by

other public and private sources. It is important that cooperation and unity be established and maintained on this program if it is to receive the future support that is so urgently needed.

A strong institutional framework in the EPA and an imaginative, effective leadership which taps the considerable talent of the several agencies involved is required to facilitate policy formulation, scientific research, and technological development to assess and preserve indoor air quality. There is an urgent need for this nation to recognize the public health significance of indoor environments to human exposures and to make an unreserved commitment to this assessment.

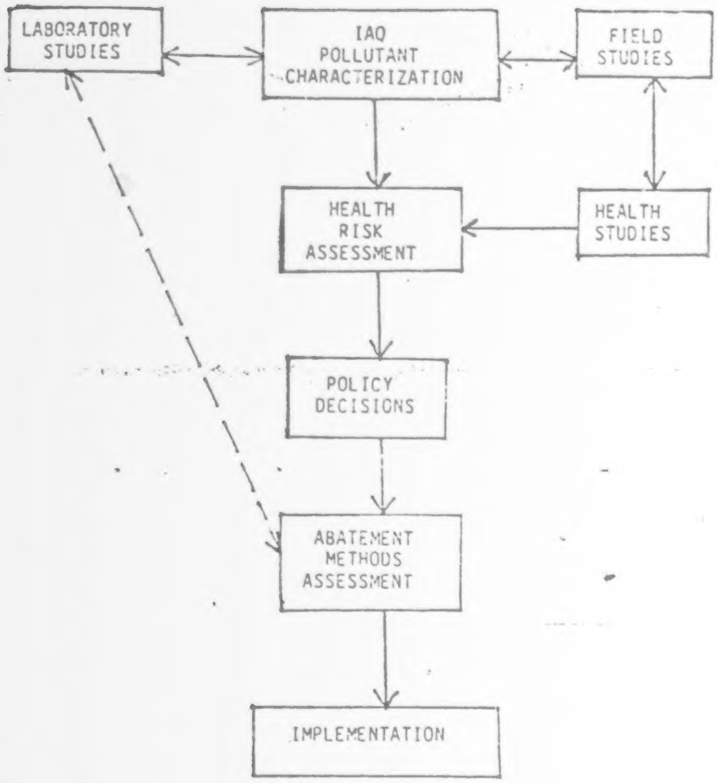

LABORATORY STUDIES
IAQ POLLUTANT CHARACTERIZATION
FIELD STUDIES
HEALTH RISK ASSESSMENT
HEALTH STUDIES
POLICY DECISIONS
ABATEMENT METHODS ASSESSMENT
IMPLEMENTATION

Figure 1. Organization Framework for Indoor Air Quality Research and Assessment

Participants in the EPA Indoor Air Quality Review Workshop
December 8 and 9, 1983
Harvard School of Public Health - Cambridge, MA
Organizers - David Harlos and John Spengler

Scott Baker (4)
US EPA
401 M Street, S.W.
Washington, D.C. 20460
202-382-7450

David Berg (3)
US EPA/OEET
401 M Street, S.W.
Washington, D.C. 20460
202-382-2602

Irwin Billick (2)
GRI
8600 Bryn Mawr Ave.
Chicago, IL 60631
312-399-8304

Robert Black (2)
EPRI
3412 Hillview Drive
Palo Alto, CA 94303
415-855-2735

Steve Bromberg (4)
US EPA/EMSL
RTP, NC 27711
919-541-2919

Sam Butcher (2)
Chemistry Department
Bowdoin College
Brunswick, ME 04011
207-725-8731 x617

William Cain (2)
Pierce Lab
Yale University
290 Congress Ave.
New Haven, CT 06519
203-562-9901

John Core (2)
Manager Air Quality Group
Nero and Assoc., Inc.
520 SW 6th Ave. Suite 1120
Portland, Oregon 97204
503-223-4150

Cliff Davidson (2)
Dept. Civil Engineering
Carnegie-Mellon University
Schenley Park
Pittsburgh, PA 15213
412-578-2951

Russel Dietz (2)
Dept. of Energy & Environment
Brookhaven National Lab
Upton. NY 11973
516-282-3059

Salvatore DiNardi (2)
School of Health Sciences
Div. of Public Health
University of Massachusetts
Amherst, MA 01002
413-545-8277

Donald Ehreth (3)
US EPA
ORD, MD-681
401 M Street, S.W.
Washington, D.C. 20460
202-382-2600

David Fine (1)
ThermoElectron, Inc.
101 First Avenue
Waltham, MA 02154
617-890-8700 x216

John Carlton Foss (2)
Human-Tech. Systems
Box 151
Lincoln Center, MA 01773
617-891-3050

Don Frankel (2)
Aerodyne Research, Inc.
45 Manning Road
Billerica, MA 01821
617-663-9500

(1)- Co-Chairs
(2)- Panel Members
(3)- EPA Presenters
(4)- EPA Observers

Participants in the EPA Indoor Air Quality Review Workshop - p.2

Carl Gerber (4)
US EPA/ORD/OEET
401 M Street, S.W.
Washington, D.C. 20460
202-382-2600

B.F. Gilmartin (2)
National Inst. Bldg. Sci.
IAQ Committee
900 17th Street, N.W.
Suite 508
Washington, D.C. 20006
202-296-3296

John Girman (1)
L.B.L.
University of California
1 Cyclotron Rd.
Berkeley, CA 94720
415-486-4668

Thad Godish (2)
Dept. Natural Resources
Ball State University
Muncie, Indiana 47306
317-285-1302

David Grimsrud (1)
L.B.L.
University of California
1 Cyclotron Rd.
Berkeley, CA 94720
415-486-4023

Richard Guimond (4)
US EPA
Office of Radiation Programs
1921 Jefferson Davis Highway, Rm 1015
Arlington, VA 22202
703-557-0704

Kalish Gupta (2)
US CPSC
Room 714
Westwood Towers Building
Bethesda, MD 20207
301-492-6837 (6994)

John Harley (2)
Director of Council
NCRP
7910 Woodmont Ave.
Bethesda, MD 20014
201-798-3705

David Harlos (Organizer)
HSPH/ESP
677 Huntington Ave.
Boston, MA 02115
617-732-1946

David Harrje (2)
CEES
Princeton University
Princeton, NJ 08544
609-452-5190

Ty Hartwell (2)
Research Triangle Institute
P.O. Box 12174
RTP, NC 27709
919-541-6453

Dale Hattis (2)
Principal Research Associate
Center for Policy Alternatives
MIT, E40 233
One Amherst Street
Cambridge, MA 02139
617-253-6468

Thomas Hauser (3)
US EPA
Director, EMSL
RTP, NC 27711
919-541-2106 (2266)

Alan Hawthorne (2)
Instrument & Measurement Group
Health & Safety Res. Division
Oak Ridge National Labs
Oak Ridge, Tennessee 37830
615-574-6246

Tom Hess (2)
Physics Department
University of Maine
Orono, ME 04473
207-581-1018

Betsy Horne (4)
US EPA Region I
John F. Kennedy Building
Boston, MA 02203
617-223-4869

Participants in the EPA Indoor Air Quality Review Workshop - p.3

Jim Hornig(2)
Environmental Studies Program
Dartmouth College
103 Nathan Smith Building
Hanover, NH 03755
603-646-2033

Robert Imhoff (2)
TVA
217 CEB
Muscle Shoals, AL 35660
205-386-2788

Mel Kollander (4)
US EPA/OPA
401 M Street, S.W.
Washington, D.C. 20460
202-382-2734

Brian Leaderer (1)
Pierce Foundation
Yale University
290 Congress Ave.
New Haven, CT 06519
203-562-9901

Michael Lebowitz (1)
Div. Repiratory Science
Westend Lab
Arizona Health Science Center
University of Arizona
Tucson, AZ 85724
602-626-6379

Robert Lewis (4)
US EPA
EMSL
RTP, NC 27711
919-541-3065

Stanley Lichtman (4)
US EPA/Rad. Prog.
1921 Jefferson Davis Highway
Room 1024
Arlington, VA 22202
703-557-3927

Pedro Lilienfeld (2)
GCA, Inc.
Technology Division
213 Burlington Rd.
Bedford, MA 01730
617-275-5444

Charles Lincoln (4)
US EPA Regional
JFK Building
Boston, MA 02203
617-223-0585

Paul Lioy (2)
Institute of Environmental Medicine
N.Y.U. Medical Center
Box 817
Tuxedo, NY 10987
914-351-5277

David Mage (4)
US EPA
EMSL/LV MD-56
RTP, NC 27711
919-541-3184

Thomas Matthews(2)
Oak Ridge National Labs
Health & Safety Research Division
Oak Ridge, Tennessee 37830
615-574-6248

Hugh McKinnon (4)
US EPA
ORD
401 M Street, S.W.
Washington, D.C. 20460
202-382-5893

Demetrius Moschandreas(1)
IITRI
10 West 35th Street
Chicago, IL 60616
312-567-4301

Brian Murphy(2)
101 Avalon Road
Newton, MA 02158
617-244-8508

A.M. Natkin(2)
Manager
Office of Environmental Affairs
EXXON Corporation
1251 Ave. of Americas
NY, NY 10020
212-398-2722 (2129)

Participants in the EPA Indoor Air Quality Review Workshop - p.4

Anthony Nero (1)
Bldg. Ventilation & IAQ Prog.
Building 90-Room 3058
Lawrence Berkeley Labs
Berkeley, CA 94720
415-486-6377

Ian Nitschke (2)
W.S. Fleming & Assoc.
5802 Court St. Rd.
Syracuse, NY 13206
315- 437-1780

Hugh O'Neil (1)
IITRI
10 West 35th Street
Chicago, IL 60616
312-567-4267

Wayne Ott (4)
US EPA
RD 680
401 M Street, N.W.
Washington, D.C. 20460
202-382-5793

Edo Pellizari (2)
Research Triangle Institute
Analytical & Chemical Sciences
PO Box 12194
RTP, NC 27709
919-541-6579

Everett (Bill) Plyer (4)
US EPA
IERL
RTP, NC 27711
919-541-2918

Jim Quackenboss (2)
502 N. Walnut Street
Madison, Wisconsin 53705
608-263-6928

Harvey Rector (2)
Geomet Inc.
1801 Research Rd.
Rockville, MD 20850
301-424-9133

Courtney Riordan (4)
US EPA
401 M Street, S.W.
Washington, D.C. 20460

Susan Rose (2)
HHA Division
ER-73
Office of Health & Environ. Researc
US DOE, GTN
Washington, D.C. 20545
301-353-5355

Marv Rosenstein (4)
US EPA/Regional
John F. Kennedy Building
Boston, MA 02203
617-587-7272

Harvey Sachs (2)
Technical Director
National Indoor Environ. Institute
5200 Butler Pike
Plymouth Meeting, PA 19462
215-825-6000

David Sanchez (4)
US EPA
IERL
RTP, NC 27711
919-541-2918

N.W. Smallwood (2)
National Inst. Building Science
IAQ Committee
G-71
PO Box 415
Rte. 16
Granville, OH 43023
614-587-7272

Kirk Smith (2)
Resource Systems Inst.
East-West Center
1777 East-West Roac
Honolulu, HI 96848
808-944-7519

John Spengler (Organizer)
HSPH
Building 1, Room 1305
665 Huntington Ave.
Boston, MA 02115
617-732-1255

T.M. Spittler (4)
US EPA/Regional Lab
60 Westview Street
Lexington, MA 02173
617-861-6700

Donald Stedman (2)
Dept. of Chemistry
University of Denver
Denver, CO 80208
303-753-3530

Tom Stock (2)
University of Texas
SPH
Health Science Center
PO 20186
Houston, TX 77225
713-792-4431

Jan Stolwijk (1)
Yale University School of Medicine
J.B. Pierce Foundation Lab
New Haven, CT 06510
203-785-2867

Gene Tucker (4)
US EPA
IERL
RTP, NC 27711
919-541-2745

Lance Wallace (3)
US EPA
401 M Street, S.W.
Washington, D.C. 20460
(202) 382-5798

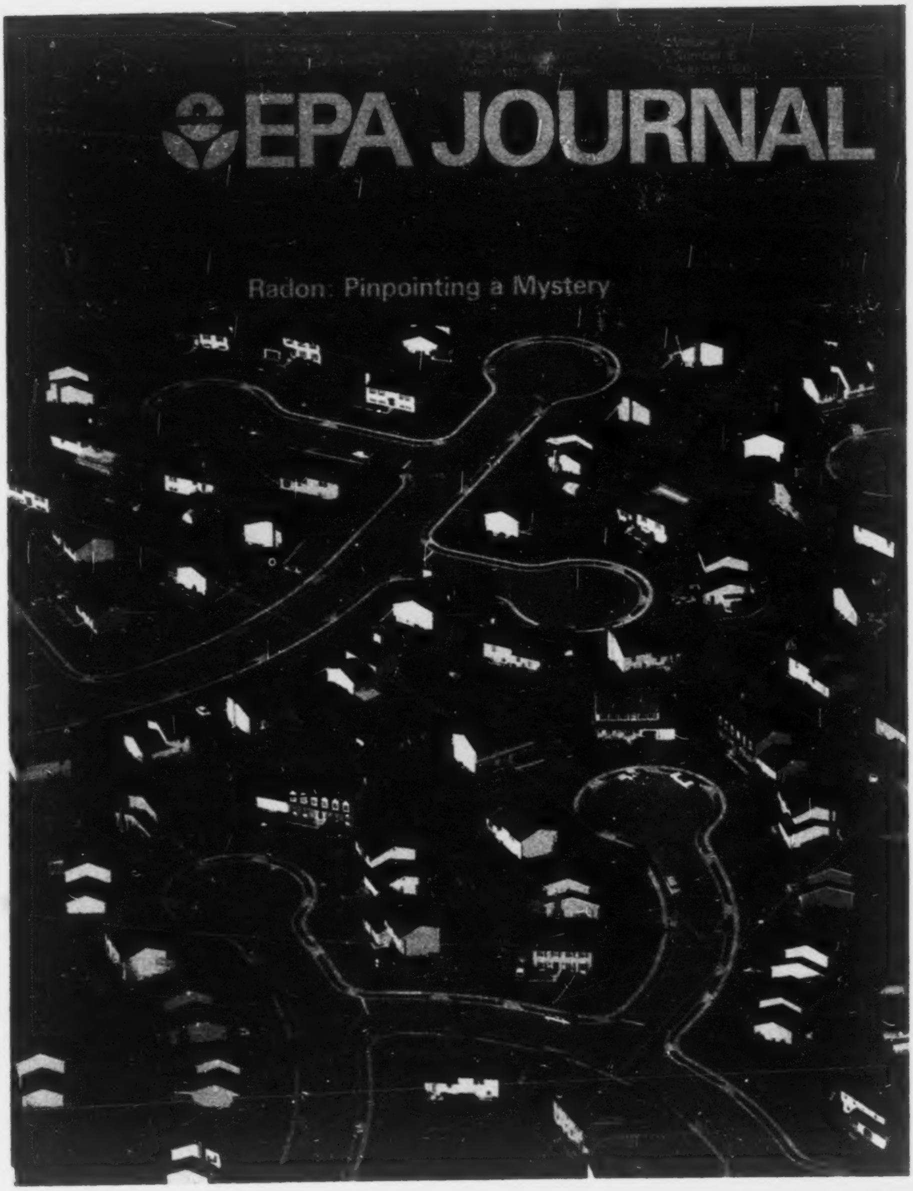
Charles Weschler (2)
Room 4G-628
Bell Laboratories
Crawford Corner Road
Holmdel, NJ 07733
201-949-7853

Dwain Winters (3)
EPA/OAR
401 M Street, S.W.
Washington, D.C. 20460
202-382-7425

Mary Lynn Woebkenberg (2)
Mail Stop R-8
NIOSH
4674 Columbia Parkway
Cincinnati, OH 45226
513-684-4266

Cary Young (2)
EPRI
3412 Hillview Drive
PO Box 10412
Palo Alto, CA 94303
415-855-2724

Larry Kirsch (2)
University of Bridgeport Law School
303 University Ave.
Bridgeport, CT 06601
203-576-4782



EPA JOURNAL

Radon: Pinpointing a Mystery

Top: The home of Stanley Watras and his family in Boyertown, PA. Discovery of high radon levels in this home has activated a national examination of the radon problem. Bottom: Mr. and Mrs Stanley Watras attend a press conference in Pottstown, PA, in April 1985. Philadelphia Electric Co. and the Pennsylvania Department of Environmental Resources announced at the conference that they would cooperate to radon-proof the Watras home.

Radon: Pinpointing a Mystery

All pollution isn't man-made. EPA's experience with the colorless, odorless gas, radon, demonstrates that fact. This issue of *EPA Journal* includes articles on the radon situation.

The magazine leads off its report with a brief explanation of the radon problem. The Agency's Deputy Administrator, A. James Barnes, discusses strategies to deal with this unusual, nonregulatory challenge. The specifics of EPA's Radon Action Program are spelled out by Richard J. Guimond, director of the Agency's efforts to deal with radon.

Pennsylvania's 18-month battle against a radon threat is chronicled by Nicholas DeBenedictis, Secretary of the state's Department of Environmental Resources. The story of how one television series focused attention on the radon problem is related by Roberta Baskin, a reporter for WJLA-TV in Washington, DC.

The personal experience of an EPA Region 3 official working directly with people who have high radon levels in their homes is described. Excerpts from EPA guidance to homeowners about radon are featured.

In a related story, Congresswoman Claudine Schneider (R-RI) argues that indoor air pollution is putting an increasing burden on the modern home.

Other stories discuss EPA's role in answering questions that emerged following the accident at the Chernobyl nuclear power plant, and asbestos in the home.

The issue concludes with two features—Update and Appointments/Awards. □

United States
Environmental Protection
Agency

Office of
Public Affairs (A-107)
Washington DC 20460

Volume 12
Number 6
August 1986

EPA JOURNAL

Lee M. Thomas, Administrator
Jennifer Joy Wilson, Assistant Administrator for External Affairs
Linda Wilson Reed, Director, Office of Public Affairs

John Heritage, Editor
Susan Tejada, Associate Editor
Jack Lewis, Assistant Editor
Margherita Pryor, Contributing Editor

EPA is charged by Congress to protect the nation's land, air, and water systems. Under a mandate of national environmental laws, the agency strives to formulate and implement actions which lead to a compatible balance between human activities and the ability of natural systems to support and nurture life.

The *EPA Journal* is published by the U.S. Environmental Protection Agency. The Administrator of EPA has determined that the publication of this periodical is necessary in the transaction of the public business required by law of this agency. Use of funds for printing this periodical has been approved by the Director of the Office of Management and Budget. Views expressed by authors do not necessarily reflect EPA policy. Contributions and inquiries should be addressed to the Editor (A-107), Waterside Mall, 401 M St., S.W., Washington, DC 20460. No permission necessary to reproduce contents except copyrighted photos and other materials.

The Radon Problem: An Overview 2

A Nonregulatory Challenge
by A. James Barnes 3

Indoor Radon: The Federal Approach
by Richard J. Guimond 5

Manning the Radon Front in Pennsylvania
by Nicholas DeBenedictis 6

Making Sense of Radon for the News
by Roberta Baskin 8

Beginning with a Phone Call
by Michael J. Chern 10

Guidance for Dealing with Radon 12

The Indoor Pollution Burden
by Claudine Schneider 14

Answering Questions About Chernobyl
by Roy Popkin 16

Advice on Asbestos in the Home
by Dave Ryan 21

Update 23

Appointments/Awards 24

Front Cover: Homes on an American landscape. Discovery of high radon levels in some homes has been a surprise. Photo by Skip Brown for Folio, Inc.

Design Credits:
Robert Flanagan,
Ron Farrah

The annual rate for subscribers in the U.S. for the *EPA Journal* is $20.00. The charge to subscribers in foreign countries is $25.00 a year. The price of a single copy of the *EPA Journal* is $2.00 in this country and $2.50 if sent to a foreign country. Prices include mail costs. Subscriptions to the *EPA Journal* as well as to other federal government magazines are handled only by the U.S. Government Printing Office. Anyone wishing to subscribe to the *EPA Journal* should fill in the form at right and enclose a check or money order payable to the Superintendent of Documents. The requests should be mailed to: Superintendent of Documents, GPO, Washington, DC 20402

EPA Journal Subscriptions

Name - First, Last PLEASE PRINT

Company Name or Additional Address Line

Street Address

City

State

Zip Code

☐ Payment enclosed (Make checks payable to Superintendent of Documents)

☐ Charge to my Deposit Account No.

The Radon Problem: An Overview

EPA has traditionally been concerned with man-made pollutants—smog, toxic chemicals, misapplied pesticides, contaminated water, and abandoned hazardous waste dumps. Now it seems we have still another problem to worry about, namely, a colorless, odorless, completely imperceptible gas, radon, generated from the natural radioactive decay of radium. Radium can be found in ordinary topsoil all around the country, but, like uranium, its parent element, it also concentrates in granite and black shale.

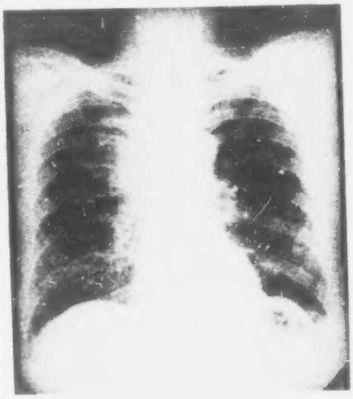
When radon gas is released it percolates up through the earth into the atmosphere, where it is thought to dissipate innocuously. However, it can also find its way into and concentrate in dwelling places through cracks in foundations, wells, drainpipes, and cinderblock walls. As radon decays, radioactive byproducts are formed and attach themselves electrostatically to dust particles in the air. These particles emit ionizing energy that can damage lung tissue and produce cancer. The problem is often less severe in schools and commercial buildings, which are usually designed for high rates of exchange between inside and outside air.

No one knows exactly how many homes in America may have seriously elevated levels of indoor radon. The Argonne National Laboratory thinks 5-10 percent of homes are contaminated. EPA believes that from one to five million private residences may be impacted. The reason for the wide variance in estimates is that the danger cannot simply be calculated by looking for uranium or radium-bearing rocks and then assuming that everyone living on the surface above them is at risk. As we have seen in eastern Pennsylvania, one house in a given neighborhood may be heavily contaminated because it sits on porous soil, while another two doors away may be well within the acceptable range of risk for an entire lifetime of exposure because it rests on a bed of clay. But many other variables may also account for these differences. Ironically, energy conservation attempts, such as caulking and insulating, may contribute somewhat to the problem by slowing air exchange rates.

It is not clear as yet how many additional cases of cancer may be attributed to radon. Estimates have run from 5,000 per year to as high as 30,000. Most experts say that radon is a leading or even *the* leading cause of lung cancer among nonsmokers. EPA plans to conduct a survey to determine the true extent of the risk. If we can predict which locations are at greatest hazard, then the public can be warned away from those sites, or houses can be built with air control systems to ensure that radon does not accumulate to intolerable levels. Such measures should be inexpensive if incorporated into a home while it is under construction, but retroactive measures can be costly.

This issue of the Journal describes the federal role in addressing the radon challenge, includes an article from a state with radon problems, reports on a media experience in making the radon problem understandable to the public, features an expert's front line experience in dealing with radon, and provides tips to homeowners to help them understand radon and what they can do about it.

EPA is working vigorously to put an effective radon program in place. Such a program cannot be created overnight, but with an appropriate investment of time and management effort, the Agency expects to make substantial progress over the coming months. □



X-ray showing diseased tissue due to cancer of the lung. Next to smoking, radon exposure may be one of the leading contributors to lung cancer in the U.S.

A Nonregulatory Challenge

by A. James Barnes

When Stanley Watras of Boyertown, PA, tripped the radiation monitor going into work at a nuclear power plant in 1984, he did more than set off lights and horns there. He also triggered alarm within the scientific and regulatory community.

Investigators discovered that Watras' home was being contaminated by radioactivity from natural, radon-bearing rock formations known as the Reading Prong. The radon levels were so high that Watras was clearly safer at work in a nuclear power plant than at home asleep in his own bed.

Radon is not a new problem. Early studies showed that radon could cause lung cancer and other health problems in miners, and it was also known that private homes could be contaminated in certain instances. For example, the use of byproducts from Western uranium and phosphate mining in construction often resulted in radon contemination in private houses.

But until Watras' experience, we had no idea that radon posed a threat to the population at large. Further studies now show that radon contamination may be a problem in many parts of the country.

Health experts estimate that radon could contribute to or cause anywhere from 5,000 to 20,000 cases of lung cancer every year. That's around 16 percent of all known lung cancers in the United States. After smoking, in fact, radon exposure may be one of the leading contributors to lung cancer. Clearly, indoor radon has the potential for being an enormous environmental health problem, and one that would require a unique approach.

Generally, EPA addresses new environmental problems either by issuing regulations or by helping states meet regulatory responsibilities. The indoor radon problem, however, does not lend itself to a regulatory approach.

(Barnes is the Deputy Administrator of EPA.)

A new home under construction. Building techniques now being developed may minimize indoor radon levels in the future.

National Association of Home Builders

First of all, radon is a naturally occurring substance. It unmistakably poses a risk, but a blameless risk. There is no one at whom we can point an accusatory finger and say, "You did this, now you fix it."

Another feature inhibiting a regulatory approach is the diversity of the radon problem. Radon levels vary from region to region, even from home to home. They depend on a building's location, style of construction, and air-tightness, as well as the amount of radon beneath it, and numerous other factors. The Watras family, for example, was exposed to radiation levels equal to about 200,000 chest X-rays a year, while radon levels in the house right next door were normal. By contrast, outdoor air pollution is shared evenly by everyone in a particular area.

The situation poses an exceptional public health issue. We now know that radon represents one of the more serious health threats facing the American public today. And we are convinced that EPA has a role to play—but we don't see it as a regulatory one. Instead, we've worked out a unique partnership with the state and local governments—unique in the sense that we are not merely cooperating, consulting, or even collaborating with the other governments. Rather, we are working in a *true* partnership with them, where they perform certain functions and we perform others. We have several levels of government working hand in hand to jointly address a problem.

We believe EPA's knowledge and specialized abilities can complement local efforts. For instance, EPA has provided survey equipment and personnel to help take measurements in the Reading Prong area. But the states retain actual responsibility for the surveys and for follow-up. We are also training state and federal personnel to diagnose and recommend remedies. But, other than for experimental mitigation projects, the federal government will not do the actual work.

Several other agencies, including the Department of Energy, the Centers for Disease Control, the U.S. Geological Survey, and the Department of Housing and Urban Development, have capabilities and expertise to contribute, too. We are working closely with them to build a comprehensive federal approach.

But while EPA will help in assessing radon hazards, demonstrating remedial techniques, and coordinating abatement efforts, perhaps our most important challenge is appropriately communicating radon risks and what can be done about them.

Our overall goal is to alleviate the potential threat that radon poses to millions of Americans. Since we're taking a nonregulatory approach to that goal, we must depend on the public to act on its own behalf. But first it needs information. The public has to know there is a threat, how large that threat may be, and how that threat can be lessened.

We at the Environmental Protection Agency must help communicate that information as accurately, honestly, and understandably as possible. We must let people know what risk radon poses to them and what they can do about it. Then we must leave the decision up to them.

It's a fine line we have to tread. On one hand, we don't want to alarm people unduly or produce stress and anxiety that could in itself be damaging to their health. On the other hand, we do believe radon is a significant hazard to public health.

If we do our job well, people will have enough information to take the vital first step of having their homes tested, where there's reason to suspect radon problems. Our information will also help them judge the risks and decide for themselves what they will do to lessen those risks. We're not going to pay for the work, but we will help inform people what options they have.

In a sense, our entire radon strategy is a means toward this end.

We're working on ways of standardizing measurement procedures and of providing quality assurance programs, so that we all speak the same language, so a reading taken in New Jersey means the same thing in California. We're working on surveys and epidemiological studies to tell us what and how much of a hazard radon actually poses to human health. And our geological studies help us pinpoint the high-risk areas of the country.

We must let people know what risk radon poses to them and what they can do about it.

But we don't feel it's enough to just point out a danger; we want to offer some solutions. We want to let people know that there are steps they can take to lessen indoor radon concentrations and what those steps are. That's where the second aspect of our approach comes in. We are conducting a program in Boyertown, PA, Clinton, NJ, and other areas to demonstrate ways of reducing radon levels in houses. The experience we gain from this program can be applied throughout the country. We are also working with the states and the housing industry to develop techniques of new home construction that might minimize radon levels in the future.

Finally, we are pursuing what we call "Capabilities Development." As the name implies, this is an effort to help local governments and industry groups develop the expertise to handle the problem themselves.

Together with some of our regional offices and the states, we are designing a program to train federal and state employees to diagnose radon problems and give homeowners proper information on remedial actions. We're also working with those who manufacture radon measurement devices, urging them to enter the residential market, and with those who make heat exchangers and air cleaners to encourage them to test their products properly, so that homeowners can select devices that are effective in reducing health risks.

In a nutshell, we are all learning what we can about radon and are jointly taking steps to make sure that knowledge is presented to the public. We'll do that with brochures, public service announcements on radio and TV, and with a special videotape made available for community groups and other interested parties.

We are confident that the extraordinary state/federal partnership we've formed will enable us to effectively communicate the danger of indoor radon to the public. We're also confident that, armed with accurate, timely, and appropriate information, people will make informed decisions.

Indoor Radon: The Federal Approach

by Richard J. Guimond

In September 1985, EPA Administrator Lee M. Thomas created a Radon Action Program to assist the states in dealing with radon problems in homes. Activities included in the EPA program can be grouped into four general categories:

Problem Assessment: EPA plans to conduct a national survey to evaluate the distribution of indoor radon levels across the country. In addition, EPA will provide technical assistance to states for surveys designed to identify specific areas that have a potential for significantly elevated levels of radon. To ensure that radon measurements are comparable and accurate, EPA has issued standardized measurement protocols and established a measurement proficiency program open to both governmental and private organizations.

Mitigation and Prevention: In this area, EPA is addressing the need for technology that is effective and inexpensive. The program includes demonstrations and evaluations of techniques to reduce radon levels in existing homes and identification and evaluation of ways to prevent radon problems from occurring in new homes.

Capability Development: The Radon Action Program includes efforts to help states and the private sector develop the technical capabilities needed: number one, to assess radon problems in homes and, number two, to help people reduce high radon levels.

Public Information: EPA is developing materials which provide information and guidance for citizens: to help them understand how to have measurements made, how to evaluate the health risks associated with high radon levels, and how to reduce those levels.

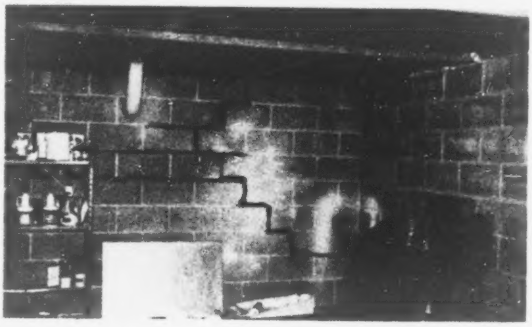
Indoor radon is too broad an issue to be addressed by any one agency. Many state and federal agencies are involved, as is a variety of private sector organizations. EPA recognizes that a coordinated approach to the problem must be taken and has designed the Radon Action Program as a partnership among its regional offices, the states, and the private sector, as well as other federal agencies.

Within the federal government, a number of agencies are participating in joint activities to address radon problems from a variety of perspectives. The primary vehicle to coordinate research efforts among these agencies is the Committee on Indoor Air Quality's Radon Work Group. The members include representatives from the Department of Energy, the Department of Housing and Urban Development, the Centers for Disease Control, the U.S. Geological Survey, the Tennessee Valley Authority, the National Institutes of Health, and the National Bureau of Standards. Participants plan and develop projects, prepare and review public information documents, and are developing a joint research strategy.

Another group which contributes to the federal effort on indoor radon is the Committee for Inter-agency Radiation Research and Policy Coordination. Through this committee, federal agencies are able to maintain a dialogue on overall research needs and long-range policy for radon-related activities.

Only through a cooperative effort involving many agencies working together can the problem of indoor radon be addressed successfully. EPA's Radon Action Program, as well as the activities of other federal agencies, helps to ensure that the radon problem is dealt with in the most thorough and effective way possible. □

To prevent radon entry, pipe (at top of picture) draws radon-containing soil gas from wall and vents it outdoors by means of an outside fan. Sealing up cracks in wall and top row of blocks enables fan to draw suction. The work in this Boyertown, PA, basement was done as part of an EPA program to demonstrate and evaluate radon reduction techniques.



(Guimond is the director of EPA's effort to help deal with radon.)

Manning the Radon Front in Pennsylvania

by Nicholas DeBenedictis

Pennsylvania has just taken what is a giant step for the Commonwealth, but a small step for a nation just becoming aware of the danger of indoor radon gas.

After testing over 22,000 homes in the last 18 months for radon and finding nearly 60 percent with high levels, the Commonwealth is now providing $3 million in low-interest loans to help owners rid their homes of natural radioactive radon. The loan program may be just the first hurdle cleared in what is assuredly a long commitment to test and help all Pennsylvanians threatened by the risk of lung cancer from radon, but it is also the culmination of 18 months of discovery, excitement, expansion, exhaustion, and, finally, achievement in fighting a very new and hard-to-believe threat.

Radon is a colorless, odorless, tasteless gas that has been produced from the radioactive decay of trace amounts of uranium since time began. Normally, the radioactive gas dissipates into the atmosphere, where its more dangerous decay products, so-called radon daughters, pose no threat.

In today's well-weatherized homes, however, that normal process is interrupted: radon gets in, but it doesn't get out. The gas and its short-lived decay products build up, creating a greater risk of lung cancer the longer the exposure.

The lack of data on natural indoor radon and its health consequences has led most experts to rely on standards, known as working levels (WL), set for uranium miners. But these standards, which measure the level of activity of radon daughters in a liter of air, are very hard to explain to people whose homes are being endangered by a colorless, odorless, tasteless gas.

How much danger it poses is not yet known, but we in Pennsylvania have by necessity taken 0.02 WL as our action guideline. I say by necessity because we did not choose 0.02 WL after studied consideration, but under an urgent need to take action.

Let me explain. Eighteen months ago, in December 1984, an engineer working on a nuclear power plant under construction near Philadelphia kept setting off portal monitors. He wasn't contaminated by anything at the uncompleted plant, so tests were taken of his home in nearby Berks County.

The result was unheard of—the engineer's home was 13.5 WL, 675 times our soon-to-be-developed action guideline. The week after New Year's, I had to advise Stanley Watras and his wife and two small children to leave their home immediately, minus even their newly opened Christmas presents, which were too contaminated to take with them.

They moved to a motel, and the Department of Environmental Resources moved into the neighborhood. The level of the Watras home was too high to be an anomaly, so we started testing neighbors' homes. Some had radon problems; others had none.

We suspected the problem lay with the Reading Prong, a granite rock formation stretching from eastern Pennsylvania to New England, first noted by the U.S. Departmnt of Energy in its national search for uranium resources in the early 1970s. In fact, the Department of Energy had been funding a survey of the Prong in Pennsylvania over the last few years.

We were confronted by a problem that stretched 45 miles from Reading to the New Jersey border, was six to-eight miles wide, and included over 20,000 homes in four counties.

A major radon program was obviously not part of DER's planned initiatives, but we knew we had to react immediately with as many resources as we could muster. We started with the Watras neighbors, then called a public meeting to explain radon and offer free testing. This procedure has been repeated again and again in the last year and a half as we have moved further out into the Prong.

Initially, we had no equipment, no program, nothing except our responsibility to address the problem. EPA's Radiation Laboratory from Montgomery, AL, loaned equipment and crews to help with the testing during those first crucial weeks of 1985. DOE brought in its helicopter to search for radon "hot spots" from the air.

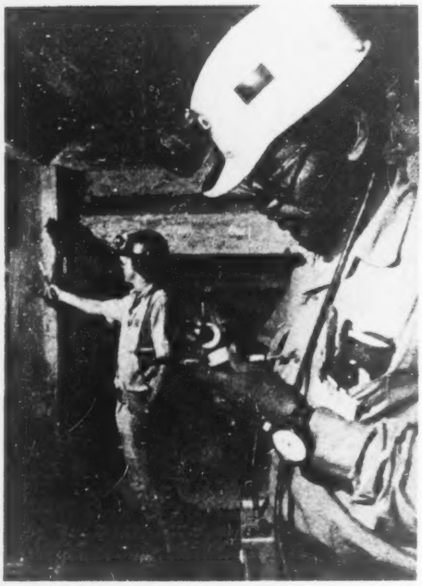
And we tested, day after day, week after week. From the beginning, we have found that a fairly constant percentage of 50 to 60 percent of the homes surveyed have radon above .02 WL.

An office was established in Gilbertsville, near the Watras home, with staff from DER's Bureau of Radiation Protection. Working from the back of a dairy store, their early struggles included just getting telephones, typewriters, and finally a computer to store the test results. Nevertheless, we were soon able to begin returning lives to normal. The Watras family, for example, was able to return home after the Philadelphia Electric Co., in cooperation with DER, had paid for remediation work by ARIX Engineers of Grand Junction, CO. This work, which cost over $32,000, involved installing a radon barrier on exterior basement foundation walls, sealing and ventilating the interior foundation wall, sealing all floor slab openings and joints, and installing a subfloor ventilation system. Radon levels in the Watras home dropped from 13.5 WL to .009 WL.

Although levels rose again last fall, adjustments, including the addition of fans, were able to bring radon in the Watras home below .02 WL.

(DeBenedictis is Secretary of the Pennsylvania Department of Environmental Resources.)

Bob Lewis, radiation health physicist with the Pennsylvania Department of Environmental Resources, unloads monitoring equipment from a van. A combination mobile field office and lab, the van serves as a sort of "mother ship" for field workers from the state's Bureau of Radiation Protection office in Gilbertsville, PA.



Workers check radon levels and ventilation in an underground mine. Estimates of the risk of lung cancer due to radon exposure are based on studies of miners.

ARIX also diagnosed 25 other radon-plagued homes for DER, and the results were collected into an advisory booklet on various building types and remedies. Residents, already unnerved by this unexpected threat, were impatient for remedies and for financial help from the government.

Our job was cut out for us when the state legislature approved $1 million to develop a testing program for the entire Prong. We had already spent nearly $1 million in unbudgeted money for testing, but had concentrated in southern Berks County where radon was initially found.

Faced with offering free radon testing to over 40,000 residences in four counties, we sought a new approach. With a press conference by Governor Dick Thornburgh and full-page, repeat advertising in five newspapers and over a dozen radio stations serving the Prong, we offered mail-in radon tests to residents in the four counties. The task was made no easier by a large Hispanic population in the Prong area that required translating all our radon materials into Spanish and hiring a bilingual community relations coordinator.

Over 20,000 testing requests came in, many in the immediate weeks after the Governor's announcement, which had also unveiled a $3 million loan program to help homeowners rid their homes of radon. Whether it was the anonymity of the mail-in testing or the light at the end of the financial tunnel, over 50 percent of all those eligible for the free testing have responded.

Radon has been a most difficult issue with which to deal. At first, Pennsylvania was the only state to know much of anything, and our basic problem was trying to communicate the risks of radon gas without raising undue panic. Now, everybody seems to know about radon and the problem is why don't we, state and federal government, know more.

We welcome this dialogue, this attention, because that's how answers are found. We think Pennsylvania has contributed and will continue to contribute as more is learned about this *national* problem called radon. But ours is not just scientific curiosity; we feel great sympathy for the many citizens whose lives have been disturbed by this unseen threat. We feel great sympathy for the parents who worry that their children may develop lung cancer in 20 years. We feel sympathy for the young homeowners who struggle to meet a mortgage each month for a home that could cost thousands of dollars more to make safe from radon.

We do not feel helpless, however. We have acted, to the extent of our state resources and to the best of our abilities. And even though the battle is just begun, we take a moment to savor conquering that first hurdle. □

Making Sense of Radon for the News

by Roberta Baskin

It's not easy to focus attention on something you can't see or smell, taste or feel, so making sense of radon for television news was a special challenge. In TV, we're at our best when there is something to show. "Covering" an invisible gas defies the imagination.

What eventually captured the media's attention was a dramatic incident. Stanley Watras of Boyertown, PA, kept setting off alarms at the nuclear power plant where he worked. When the experts finally traced the problem to fantastically high levels of radon gas in his home, the media had something tangible—a family of victims living in a radioactive cloud. That revelation led to a flurry of media attention and radon testing in the Reading Prong area, a radon-bearing geological formation that stretches through Pennsylvania. But the issue's coverage was represented as a local problem. In the nation's capital, we could pity those poor families in Boyertown without being touched by the problem directly. We were safe at home in our beds. Or so we thought.

Radiation experts seemed to agree that, as serious as the radon problem is along the Reading Prong, it is not confined to that geographic area. In fact, there seemed to be acknowledgment that, as more homes are tested nationwide, radon at even higher levels would be discovered. It left us wondering what we might find in the Washington, DC area.

Checking with the Department of Energy about that possibility was not encouraging. DOE officials felt radon testing here would be uneventful, certainly not a high priority. But, since there's no way to know without doing the tests, they decided to help us out, providing we followed their guidelines and standards. If it turned out there was no radon hazard in the Washington area, we could at least give people an added measure of peace of mind.

The ground rules DOE set up for us involved coming up with a random sample of some fifty single-family homes with basements. We would test them in the basements and living areas, first in the fall, then following up in the winter when homes are "tightened up." We came up with our volunteers in the District of Columbia, Maryland, and Virginia, and arranged to place carbon cannisters in the homes to take air samples. These were all sent to DOE's Environmental Management Laboratory in New York City for analysis.

It's not easy to focus attention on something you can't see or smell, taste or feel.

The results surprised all of us. In simple terms, the radon levels were about three times higher than the known national average. Nearly half the homes tested were above the Environmental Protection Agency's suggested action level. Although our levels didn't approach the Reading Prong problem, they were definitely cause for concern. A few of the homes had radon levels exceeding the Bureau of Mines safety standard for uranium workers. We had opened a Pandora's Box. The five months of preparation that went into our series of radon reports did not prepare us for the public's reaction.

The station received hundreds of calls from homeowners who wanted to find out how to get their homes radon-tested. But our phones weren't the only ones ringing off the hook. The state health departments in Maryland and Virginia were each getting thousands of calls. Officials complained they couldn't get their coats off, much less handle anything else but radon calls. All state resources for radiation issues were channelled into handling endless radon questions from the public. The EPA got its fair share of the spillover. And one person, WJLA-TV's sports anchor Frank Herzog, got more than his fair share of calls.

Herzog was one of the volunteers in our radon survey. He had given up smoking three years ago, but radon tests in his home revealed he was getting a dose similar to that from smoking a pack and a half of cigarettes a day. It was not cheery news, but he was a good sport about it. He was an even better sport about the calls he started getting at all kinds of hours from people who wanted to know what they should do about radon because they figured he was an expert. Fortunately, he was able to lower his home's radon levels by following some simple recommendations.

As hundreds more homes were tested, we started to get new data. Unlike the volunteers in our radon survey, many of the homeowners were eager to keep the problem quiet. In fact, the majority of callers were far more concerned about their property values than any possible health hazard. They were anxious to find out what they could do about it, and equally anxious that no one else learn of their problem. In many cases, this made it difficult to do follow-up reports. For example, one woman who called was outraged about how high her radon levels were and how she couldn't get anyone to do anything for her. Indeed they were high: 20 times higher than the EPA's

(Roberta Baskin is a reporter for WJLA-TV in Washington, DC.)

On the air: TV newswoman Roberta Baskin reports on the results of radon tests in the Washington, DC, metropolitan area.

recommended action level. She was particularly concerned because she lived in a new house and the developer was building hundreds more homes up the street from her. On her first call, she wanted the world to know about the potential hazard. By the second call, she wanted to keep it very, very quiet. She said she'd found someone to do the remedial work on her home, providing she not talk to any journalists. That case was not unique.

Occasionally we heard from families who wanted to share their experiences in the hope others would benefit. In one of those cases, the family was building a bedroom in the basement for their eight year-old son. Radon testing showed a level of radon equal to getting a chest X-ray every passing hour. Although most of the renovation was complete, the parents decided not to move their son downstairs until they found a way to bring down the radon levels. But, as a benefit of the publicity, they were contacted by an engineering company that specializes in radon-proofing homes. The company is doing the work at no charge in the hopes it can publicize its success later on.

In the aftermath of our radon reports, the Maryland and Virginia health departments have put more resources into their radon efforts. In Virginia, the state is now radon-testing about 600 homes to get a better notion of how serious the problm is there. An "800 number" was installed to help answer questions, and a booklet was produced to explain more about radon, along with basic advice about what to do about it. Both Virginia and Maryland joined EPA in encouraging homeowners to get their homes tested. We took a cue from that advice to try and persuade officials to radon-test the White House. The officials we were referred to were delighted to hear from us because they wanted to get their own homes tested. But our calls did lead to dozens of detectors being placed around the White House, all of which turned up low readings, according to a spokesman.

It was rewarding to get people in high places and near places and far away places to become aware of the radon gas problem and how it may affect them directly. The fact that dozens of television, radio, and newspaper reporters contacted us from all around the country is a hopeful sign that the story will continue to unfold everywhere. That's important since there is a temptation to ignore a problem like radon because it's unseen. Another temptation is for homeowners to cover it up. The media really have a responsibility to stop that from happening. The problem won't go away by itself. And the consequences will only grow worse. In fact, hiding the issue only raises the specter of future litigation. The developers, the builders, the home sellers who conceal a radon hazard today are likely to be the subject of tomorrow's stories. There will also be future stories about fly-by-night radon-testing companies . . . the sort preying on old ladies. The mind reels at the entrepreneurial possibilities. Mayonnaise jars could be used to test for radon, and who knows what else. But the more attention radon gets, the more enlightened the public will be, meaning the more responsibly it will be dealt with. An important role for reporters is to keep radon from being relegated to the obscurity of the basement. People need to be remindad that it's there . . . and that they *can* do something about it. □

Beginning with a Phone Call

by Michael J. Chern

"When the lady you're speaking to breaks down crying because she's worried that she and her family may die of lung cancer, you feel like dropping everything else and spending a month helping this one person until her problem is solved," says Bill Belanger, EPA Region 3's radiation expert for the last five years. Belanger is talking about his first-hand experience working directly with people who have high radon levels in their homes.

Until a year and a half ago, Belanger spent much of his time working on emergency drills for nuclear power plants. His only experience with radiation in houses was in a Lansdowne, PA, home which had been used by a university professor in the 1940s to refine radium for use in hospitals. The house is now so contaminated with radioactivity that it was evacuated under the emergency provisions of Superfund and added to the Superfund National Priorities List for remedial cleanup.

Now, with the discovery of high radon levels in the Reading Prong area of Pennsylvania, Belanger spends almost all his time on this problem.

First Word

Belanger's serious involvement with naturally occurring radon began on December 19, 1984, with a phone call from Charles Porter, Director of EPA's Eastern Environmental Radiation Facility in Montgomery, AL.

Belanger remembers that the conversation began with Porter saying, "Bill, you better sit down. There's a house in your region that has 13 working levels (WL)!" ("Working level" is one of the measures used to express radon exposure.)

Belanger replied, "Are you sure you didn't slip the decimal a couple of places?" Until that time, everyone's experience with natural radon had indicated that a high indoor reading was 0.1 WL; Belanger had taken readings of 0.3 WL in the Lansdowne house and had considered that extraordinarily high.

(Chern is a former Public Information Officer for EPA Region 3.)

Porter's 13 WL referred to radon levels found in the home of Stanley Watras of Boyertown, PA. Watras, a construction engineer, had set off radiation monitors while entering the Limerick nuclear power plant where he worked, and the source of the radiation had been identified as his house.

Early Actions

EPA's radiation experts quickly realized that the discovery of radon in the Watras house probably would mean an extensive monitoring program in the Reading Prong. "It would have been too much of a coincidence to expect that the only homeowner with high radon readings was an employee at a nuclear power plant," says Belanger. "If it was a naturally occurring problem, there had to be more houses with it."

One of the first concerns of state and federal officials in the monitoring program was whether to use protective equipment for the monitoring team. Normally, workers who knowingly go into areas with high contaminant levels wear such equipment, and homes with 10 to 20 WL are considered to have very high levels.

"But if you were a homeowner, what would you think if we showed up at your door in gas masks and said we wanted to take radon readings in your home?" asks Belanger. The EPA field staff finally decided to work without protective equipment. They reasoned that, even if they entered a few houses with high radon readings, their length of exposure would be too brief to have a significant health effect.

Working in the Reading Prong

At the request of Pennsylvania environmental officials, Belanger joined the field monitoring effort in April 1985 to take radon measurements in the Reading Prong.

The first step in measuring radon is to take a screening measurement to determine the highest level in the house. EPA recommends that this reading be taken in the basement or the lowest part of the house during the heating season or some other time when the house is closed. If this reading is low, radon can usually be dismissed as a problem.

If a high level is found, however, further measurements are taken in the most frequently occupied parts of the house. Only after these additional readings are completed is it possible to tell the extent of the radon problem.

"But it's up to the homeowner to decide what will be done," says Belanger. "We can make a recommendation as to what level the homeowner should shoot for. We have been recommending the .02 WL, but the homeowner doesn't have to accept that. He may not be willing to spend the amount of money needed to reach that level. Or, perhaps he is not satisfied that .02 WL is safe enough, and he wants to go lower. He may decide he is willing to give up regular use of his basement. All these things are alternatives that only the homeowner can and should decide."

Remediation Research

Helping homeowners decide what to do is another part of EPA's Radon Action Program. The Agency has embarked on an intensive research project in the

Hole in the floor of a house under construction is an entry point for radon. The hole—a result of standard construction practices—will probably be hidden by floor covering when the house is complete. Other common radon entry points are sump pump holes, crawl spaces, and cracks in foundation slabs. "If we can easily find the entry places of radon," says EPA expert Bill Belanger, "the control costs are usually reasonable."

Checking his watch, Bill Belanger times a five-minute indoor air sample for radon testing.

Reading Prong to try various radon reduction methods in a variety of houses representative of those found most often in the area. The project is designed to find affordable as well as effective radon reduction techniques.

According to Belanger, the costs of radon removal can vary greatly. "If we can easily find the entry places of radon, the control costs are usually reasonable," he says. "The job can often be done for a thousand dollars or less."

"On the other hand," he warns, "if the house already has a finished basement or there is nothing obvious to do, it may cost many thousands of dollars because you may have to make major structural changes."

EPA also has found that the higher the level of radon in a house, the higher the costs of control. "If you have 0.2 WL in the house and you want to get down to .02, that's a 90 percent reduction," says Belanger. "That's not too hard to do. But if you have 2 WL and want to get to .02, that's a 99 percent reduction."

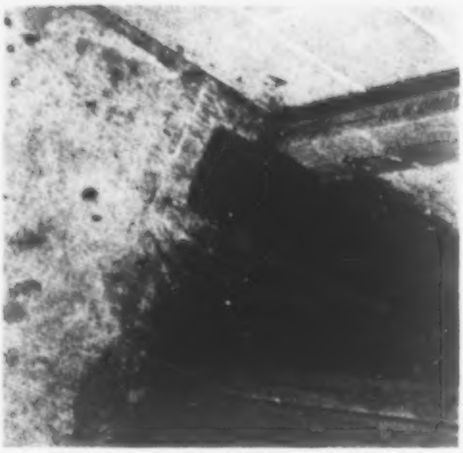
EPA has looked at many reduction techniques that reduce radon by about 90 percent. But it is difficult to predict exact costs until the construction of the house is determined. It's also difficult to be sure that control measures work all the time. Take, for example, Belanger's October 1985 visit to Boyertown, PA, with a U.S. Senator who was inspecting one of the homes in EPA's remediation research program. It turned out to be a classic example of Murphy's law that anything that can go wrong, will.

Belanger recalls that, after explaining to the Senator some of the things that had been done to the house, he was asked to take a radon measurement.

"This home originally had a concentration of 7 WL," says Belanger, "and my own measurements taken soon after the house was fixed showed only a little above 0.02 WL. But this day, I got a measurement of 3 WL!"

"All of us there—the Senator, the homeowner, and I—realized something was very wrong," Belanger continues. "Since the highest reading was previously in the basement, I went down there to take a reading. It was 15, more than twice as high as the house had been before we did anything to it."

"At this point, the homeowner was at the point of tears," says Belanger.



After the Senator left, Belanger spent some time looking around the house and found a bedroom window open on the downwind side of the house. No other windows were open, yet air was rushing out of that window.

"I figured that, since the rest of the house was sealed, the air going out must be coming in through the foundation, bringing radon with it." Belanger closed the window, went down to the basement, and opened up one of the basement windows on the upwind side, allowing fresh air to enter the house. Within an hour, the level of radon had been reduced by a factor of two.

That afternoon state officials took more measurements, and the level had dropped to under 0.1 WL. They returned the next day and took readings under .01 WL—well within the state and proposed federal guidelines.

Says Belanger of the experience, "We learned a lot that day. And not just that things can go wrong at inopportune moments. Most importantly, we learned that open windows and air currents can have a dramatic effect on radon control systems."

Working with Citizens

Despite occasional events like the Boyertown visit, Belanger has nothing but positive things to say about his experiences working with people who have radon problems.

"All of us from EPA working there have an enormous amount of sympathy for the people we meet with high radon levels in their homes," says Belanger. "We would like to help every one of them individually. But we just can't. There aren't enough of us. Our primary job must be to provide advice and assistance to the states so that they can help everyone with a problem."

The most important thing in providing help, he believes, is getting the people's trust. "They believe you and accept your help if you give it to them straight," he says.

"If you try to mislead them in any way, if you try to minimize the problem or make it sound more serious than it is, they pick it up right away. People living up there (in the Reading Prong) have spoken to others about the problem. They know the background. So, you can't throw bull at them.

"I feel very good about my work there," says Belanger. "It's like working as a medic in an ambulance. You can point to the people you've helped. This is what drives you. You feel like you're saving lives. That's the real motivation in a job like this." □

Guidance for Dealing with Radon

To help people understand radon, EPA and the U.S. Centers for Disease Control recently published A Citizen's Guide to Radon: What It Is and What to Do About It. *Following are excerpts from this booklet.*

How is radon detected?

Since you cannot see or smell radon, special equipment is needed to detect it. The two most popular, commercially-available radon detectors are the charcoal canister and the alpha track detector. Both of these devices are exposed to the air in your home for a specified period of time and sent to a laboratory for analysis.

There are other techniques—requiring operation by trained personnel—which can be used to measure radon levels, but such techniques may be more expensive.

Your measurement result will be reported to you in one of two ways. Results from devices which measure radon decay products are reported as "Working Levels" (WL). Results from devices which measure concentrations of radon gas are reported as "picocuries per liter" (pCi/l).

How can I get a radon detector?

Homeowners in some areas are being provided with detectors by their state or local government. In many areas, private firms offer radon testing. Your state radiation protection office may be able to provide you with information on the availability of detection devices or services.

The U.S. Environmental Protection Agency conducts a Radon Measurement Proficiency Program. This voluntary program allows laboratories and businesses to demonstrate their capabilities in measuring indoor radon. The names of firms participating in this program can be obtained from your state radiation protection office or from your EPA regional office.

How should radon detectors be used?

. . . have a short-term "screening" measurement made to give you an idea of the highest radon level in your home. Thus, you can find out quickly and inexpensively whether or not you have a potential radon problem.

The screening measurement should be made in the lowest livable area of your home (the basement, if you have one). All windows and doors should be closed for at least 12 hours prior to the start of the test, and kept closed as much as possible throughout the testing period . . .

Depending upon the result of your screening measurement, you may need to have follow-up measurements made to give you a better idea of the *average* radon level in your home . . .

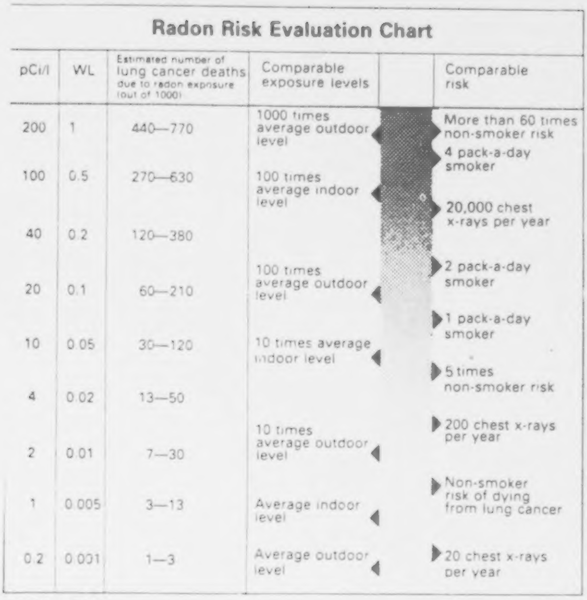
We strongly recommend that you make follow-up measurements before you make any final decisions about whether to undertake major efforts to permanently correct the problem.

Follow-up measurements should be made in at least two lived-in areas of your home. If your home has lived-in areas on more than one floor, you should make measurements in a room on each of the floors. The results of the follow-up measurements should be averaged together.

What do my test results mean?

The results of your follow-up measurements provide you with an idea of the average concentration throughout your home. The actual risk you face depends upon the amount of time you are exposed to this concentration.

One way to think about the risk associated with radon exposure is to compare it with the risk from other activities. Figure 1 gives an idea of how exposure to various radon levels over a lifetime compares to the risk of developing lung cancer from smoking and from chest x-rays. Figure 1 also compares these levels to the average indoor and outdoor radon concentrations.



Radon Risk Evaluation Chart

pCi/l	WL	Estimated number of lung cancer deaths due to radon exposure (out of 1000)	Comparable exposure levels		Comparable risk
200	1	440—770	1000 times average outdoor level		More than 60 times non-smoker risk 4 pack-a-day smoker
100	0.5	270—630	100 times average indoor level		
40	0.2	120—380			20,000 chest x-rays per year
20	0.1	60—210	100 times average outdoor level		2 pack-a-day smoker
10	0.05	30—120	10 times average indoor level		1 pack-a-day smoker
4	0.02	13—50			5 times non-smoker risk
2	0.01	7—30	10 times average outdoor level		200 chest x-rays per year
1	0.005	3—13	Average indoor level		Non-smoker risk of dying from lung cancer
0.2	0.001	1—3	Average outdoor level		20 chest x-rays per year

Figure 1

How quickly should I take action?

In considering whether and how quickly to take action based on your test results, you may find the following guidelines useful. EPA believes that you should try to permanently reduce your radon levels as much as possible. Based on currently available infromation, EPA believes that levels in most homes can be reduced to about 0.02 WL (4 pCi/l).

If your results are about 1.0 WL or higher, or **about 200 pCi/l or higher:**

Exposures in this range are among the highest observed in homes. Residents should undertake action to reduce levels as far below 1.0 WL (200 pCi/l) as possible. We recommend that you take action within several weeks. If this is not possible, you should determine, in consultation with appropriate state or local health or radiation protection officials, if temporary relocation is appropriate until the levels can be reduced.

If your results are about 0.1 to about 1.0 WL, or **about 20 to about 200 pCi/l:**

Exposures in this range are considered greatly above average for residential structures. You should undertake action to reduce levels as far below 0.1 WL (20 pCi/l) as possible. We recommend that you take action within several months.

If your results are about 0.02 to about 0.1 WL, or **about 4 pCi/l to about 20 pCi/l:**

Exposures in this range are considered above average for residential structures. You should undertake action to lower levels to about 0.02 WL (4 pCi/l) or below. We recommend that you take action within a few years, sooner if levels are at the upper end of this range.

If your results are about 0.02 WL or lower, or **about 4 pCi/l or lower:**

Exposures in this range are considered average or slightly above average for residential structures. Although exposures in this range do present some risk of lung cancer, reductions of levels this low may be difficult, and sometimes impossible, to achieve.

Remember: There is increasing urgency for action at higher concentrations of radon. The higher the radon level in your home, the faster you should take action to reduce your exposure.

How can I reduce my risk from radon?

Your risk of lung cancer from exposure to radon depends upon the amount of radon entering your home and the length of time it remains in your living areas. Listed below are some actions you might take to immediately reduce your risk from radon.

- Stop smoking and discourage smoking in your home.
- Spend less time in areas with higher concentrations of radon, such as the basement.
- Whenever practical, open all windows and turn on fans to increase the air flow into and through the house. This is especially important in the basement.
- If your home has a crawl space beneath, keep the crawl space vents on all sides of the house fully open all year.

The booklet from which the above text is excerpted is available from state radiation protection offices. Also available from these sources is another new EPA publication Radon Reduction Methods: A Homeowner's Guide, *with information on methods which might be used to reduce the level of radon in homes.*

The Indoor Pollution Burden

by Claudine Schneider

"Your home is your castle," goes the old saying, and most Americans are still convinced that home is one of the last refuges left from the many ills of modern living. Unfortunately, this belief may be far from reality as the evidence mounts regarding the harmful effects of indoor pollution.

EPA spends $230 million to control air pollution outside, but only $2 million on indoor air pollution. Based on the amount of time Americans spend in buildings, federal research spending on indoor air quality amounts to only two cents per person-year of exposure, compared to about six dollars per person-year of exposure outdoors. Yet indoor air is more polluted on the average than outdoor air, and we spend more than 75 percent of our time in it. (Infants and the elderly, the groups most sensitive to pollutants, spend 90 percent of their time indoors.) Through indoor air, we're exposed daily to known carcinogens such as asbestos, as well as to pesticides, cleaning supplies, and other chemicals whose effects are yet unknown. The tobacco smoke inhaled by active and passive smokers includes over 2,000 chemicals, many of which are known toxins and carcinogens. And now we've learned that radon—a naturally occurring radioactive gas that causes lung cancer—is present in millions of American homes at exposure levels greater than those regulated in uranium mines.

Indoor air is more polluted on the average than outdoor air.

In fact, the majority of known pollution-related deaths in the U.S. is caused by tobacco (350,000 deaths per year) and radon (5,000 to 20,000 deaths per year). Yet the government continues to subsidize tobacco farmers, allow seductive cigarette advertising, and support a reduction of the cigarette tax. The case of radon, too, is noteworthy because it could be solved relatively easily. If EPA and other concerned agencies put a reasonable emphasis on radon alone, we could prevent several thousand lung cancer deaths each year.

Techniques for identifying and measuring indoor radon have improved immensely over the past 10 years, and inexpensive monitors, sensitive and accurate enough to determine whether radon is a concern in individual homes, are now available for less than $20. Many homeowners can modify their homes fairly simply to minimize radon concentrations by closing off the radon entry points and ventilating as near to the source as possible.

Some of the best radon research and monitoring equipment has emerged from federal energy programs analyzing the ventilation conditions of energy-efficient buildings. Popular press reports have repeatedly implicated "tightened" buildings as the cause of indoor pollution, but the evidence to date does not indicate that energy conservation efforts are creating health hazards. According to a report in progress by the American Council for an Energy Efficient Economy, researchers found homes with extremely low ventilation rates where radon was not a concern, while leakier homes experienced high radon levels. The key is to identify those homes with significant sources, and implement available control techniques.

One of the problems of this health threat is that the location of homes with high radon levels remain largely unknown. A national survey of indoor radon in the U.S. housing stock is of paramount importance to understand the distribution of this hazard across the country. Additional data on the distribution of other significant indoor pollutants are also needed.

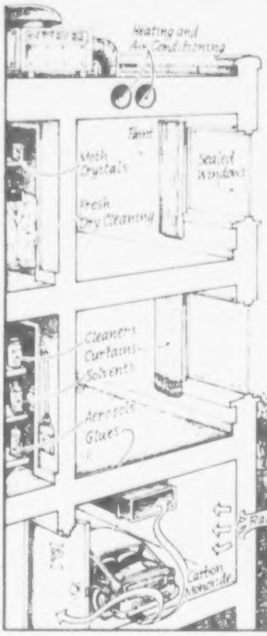
A key part of the problem lies with Congress which needs to reconsider and update the way it funds and organizes all the agencies that are protecting our health. Agency budgets should be related to their potential contribution to our well-being.

Indoor air hazards can be significantly reduced before we have a problem of epidemic proportions.

That is why I support funding specifically allocated for the study of indoor environments. This past year I sponsored the Indoor Air Quality Act of 1985, which was incorporated into EPA's pending Office of Research and Development authorization bill. It would require that EPA:

- Coordinate federal, state, local, and private research efforts relating to indoor air;
- Prepare a research plan;
- Conduct research to identify, monitor, characterize, and measure pollutants;

(Congresswoman Schneider (R-RI) is a member of the U.S. House Committee on Science and Technology where she is ranking minority member of the Subcommittee on Natural Resources, Agricultural Research and Environment. She is also a member of the House Merchant Marine and Fisheries Committee.)



Air pollution from many sources assails us in our homes and offices, as shown in this cut-away view of a dwelling.

Studies show that indoor pollution can be two to five times greater than outdoor pollution. Many of its sources are common household products.

Air Pollution, In My Home?

study the effects on health; identify high-risk building types; evaluate control technologies; and disseminate information;

- Establish broad-based advisory groups; and
- Report on the risks to human health associated with indoor air pollution, the need for further research, and the need for possible federal actions to mitigate risks associated with indoor air problems.

I also consider it appropriate for the Committee on Indoor Air Quality, which has been designated by Congress to coordinate the research efforts of the 16 agencies examining indoor environments, to convene a study with EPA to reprioritize funding within the related agencies so that each dollar spent has a fair chance of contributing equally to improving our health and well-being.

Initiatives in the private sector have also contributed to our knowledge of indoor air quality. I admire the efforts of groups such as the Consumer Federation of America (CFA) and the American Council for an Energy Efficient Economy (ACEEE) in bringing this issue to the attention of policy-makers and consumers alike. CFA has designated indoor air quality as its number one health and safety issue, and has held conferences and published a newsletter on this topic. ACEEE's ongoing research clearly indicates that healthy indoor air in energy-efficient homes is attainable, but only a national commitment to achieving this important public health goal will get us there.

Much remains to be done, particularly the establishment of an information clearinghouse to effectively disseminate information on indoor air quality. The scientific and technical communities working on these problems need to exchange information, as do the state and local health agencies. And last, but certainly not least, individual citizens who may have potential problems desperately need information.

Since I began focusing on this issue, I have received many requests for information, as I am sure EPA and other organizations have also. Consumers want to know: How do I know if I have a problem? How can I measure for pollutants in my home? What health effects are indicative of an indoor air pollution problem? What should I do to combat unsafe levels? These and scores of other questions need to be answered. Arming consumers and building operators with accurate information will help prevent the fraud and marketing abuse which lurk around the corner whenever a new hazard is coupled with a lack of adequate and reliable information.

There is another old adage I strongly believe, and that is "an ounce of prevention is worth a pound of cure." With a commitment by EPA, other government agencies, and the private sector, I believe that indoor air hazards can be significantly reduced before we have a problem of epidemic proportions and before Americans become resigned to living in homes that make them sick.

Answering Questions About Chernobyl

by Roy Popkin

Before the reactor blew on April 26, few Americans had heard of the Soviet Union's Chernobyl nuclear power plant; 48 hours later, it was a household word, the anxious focus of the press, the public, and the government. Was dangerous radioactive fallout heading to the United States? Was the health and safety of Americans abroad being threatened? No one knew.

Answering these questions became the job of the Task Force on the Soviet Nuclear Accident. At the peak of its activity, it was measuring domestic radiation levels, monitoring foreign levels, tracking the situation at Chernobyl, and handling hundreds of phone calls everyday. For almost a month, the Task Force was *the* source of information for Americans on the world's worst nuclear disaster.

Chernobyl was a secret disaster at first. The initial evidence that a major nuclear accident had occurred came not from Soviet sources, but from Sweden, where on April 27 workers at a nuclear power plant were found to have radioactive particles on their clothes. It was Sweden's search for the source of radioactivity—there was no leak at the

At EPA's Eastern Environmental Radiation Facility in Montgomery, AL, Vicki Lloyd tests samples of pasteurized dairy milk for Strontium 90, a fission product that may be present in nuclear fallout. Following the accident at Chernobyl, EPA increased milk monitoring from once a month to twice a week.

What Does a Task Force Really Do?

The letter from the White House was short and to the point. From Larry Speakes, Deputy Press Secretary to President Ronald Reagan, it was addressed to EPA Administrator Lee M Thomas:

"My congratulations on a job well done in the wake of the Chernobyl accident. The interagency group you headed so effectively ought to be used as a model for future situations like this."

Interagency task forces are not unusual in emergencies and even in relatively mundane situations. The Chernobyl task force was formed quickly, its structure based in large part on an existing Memorandum of Understanding which gave EPA the lead role when there was an atmospheric nuclear detonation abroad, as in the case of two Chinese nuclear tests which led to extensive monitoring in the United States. Although Chernobyl was not the same, the required response activities were judged to be quite similar.

The Task Force met for the first time at 5:30 PM on Wednesday, April 30. The fire in the graphite core was still burning and the situation at the reactor site was still unclear. The Task Force's first job was to assign tasks:

EPA would be the clearinghouse for offers of assistance to the Soviet Union and would coordinate with the Department of State; DOE would help

(Popkin is a writer/editor for the EPA Office of Public Affairs.)

Swedish plant—that led to the first hint of a nuclear problem in the Soviet Union.

EPA first learned about a possible radiological incident from press and citizen inquiries coming in on Monday, April 28. The Agency's Press, Radiation, and International Activities offices began fielding calls while working with the State Department, the Nuclear Regulatory Commission (NRC), and the Department of Energy (DOE) to find out what was happening. Although the Soviet news agency, TASS, finally issued a terse statement that evening confirming an accident at the Chernobyl plant, the Soviets offered no details. The resulting information vacuum fueled rumors of all kinds, from fatality estimates to speculation about fires in adjoining reactors.

Tuesday morning, an interagency group met at the White House to review what little information was then available. Although President Reagan was en route to the economic summit meetings in Tokyo, EPA was confirmed as the "lead" agency for coordinating the federal response, and EPA Administrator Lee Thomas was designated head of the Task Force. In addition to EPA, the Task Force was to include DOE and NRC, the White House, the Departments of State, Interior, and Agriculture, the National Oceanic and Atmospheric Administration (NOAA), the U.S. Air Force, the Food and Drug Administration (FDA), the Federal Emergency Management Agency (FEMA), the Federal Aviation Administration (FAA), and the U.S. Public Health Service.

The Task Force immediately established an "up front" approach to

the FAA take measurements; the Centers for Disease Control/FDA medical network—normally used in drug-tampering incidents—would be used to inform state health officers. The lead for public information would be EPA, and DOE would handle congressional liaison. At the same meeting, the Task Force also decided to step up the monthly ERAMS milk monitoring to twice a week.

On Thursday, the Task Force broadened its assignments:

- The State Department was to report on the Soviet obligation to report data.
- EPA's Office of International Activities was to make recommendations on an international information exchange.
- EPA's Office of Radiation Programs (ORP) was to work with the Department of State to prepare a cable requesting technical information from the USSR and to solicit information based on questions submitted by Task Force agencies.
- A Health Working Group was formed to examine potential long- and short-term health effects, identify symptoms and effects, and distribute information to health officials.
- ORP/EPA was to be responsible for day-to-day events, reporting, and data collection.
- A DOE-NRC-FEMA-CIA subgroup was to develop and evaluate possible reactor scenarios.
- NOAA was to provide the meteorological and dispersion information for the daily Task Force report.
- DOE was to evaluate the technical aspects of extinguishing a graphite reactor fire.

The Task Force met daily through May 9, then skipped the weekend, although updated task force reports were issued by the EPA press office on Saturday and Sunday. Excerpts from minutes of Task Force meetings show the variety of actions taken by the interagency group.

May 2—The Task Force decided to contact counterpart agencies in affected countries to obtain radiological data. (NRC placed calls to 18 countries.)

May 3—NRC sent a notice to its licensees requesting they report any unusual radiation levels. EPA was to get radiological data from DOD bases in Europe and Japan, as well as coordinate data from several other countries.

NOAA reported that the radioactive plume had reached Japan, but no numbers were firmly established. The Department of State was to call our embassy there, and EPA was to determine when data from the military would be available. State and HHS were to work on another cable to offer medical liaison with the USSR. USDA reported that the World Agriculture Outlook Board had requested data from the Task Force to consider in its grain and sugar beet projections. Early radiation readings did not appear alarming.

May 6—The Task Force decided to begin publishing consolidated data, with positive U.S. measurements placed in context, their meaning and health implications explained. The Health and Agriculture Working Group(HAWG) reported on projected health effects and identified Protective Action Guides. EPA was asked to provide radiation data on returning U.S. citizens to the Health and Agriculture group. Chairman Thomas directed that any discussions should explain protective action guides and compare them with ERAMS and other U.S data.

May 12—The Health and Agriculture Working Group reported it had completed development of an advisory outlining levels of concern for imported products.

May 14—the last meeting of the Task Force. The group decided that EPA would continue its operations, including public reports and intensified monitoring until deemed to be not necessary. Other agencies would continue to cooperate as needed. The Task Force would issue a summary health and dose assessment report with EPA serving as the lead agency. EPA and other agencies were to evaluate the lessons learned in the context of their individual responsibilities and push to modify their own procedures accordingly. HAWG would submit to Lee Thomas a list of areas needing improvement. The scope of the Memorandum of Understanding was to be re-examined, as well as the need for real time dose and health information, and the role of the State Department, especially in relation to the need for an international capability for faster, real-time data collection. Thomas announced he intended to send a wrap-up memorandum to the White House. □

the news media, providing access to such top level experts as Harold Denton, Director of Nuclear Reactor Regulation for NRC; Dr. Lester Machta, director of NOAA's Air Resources Laboratory; Dale Bunch, DOE Deputy Assistant Secretary for Reactor Deployment; and Sheldon Meyers, director of EPA's Office of Radiation Programs (ORP) and head of Task Force support efforts (with a quickly mobilized team of ORP staff serving as the focal point for Task Force support). At the first press conference, Thomas promised every effort to provide as much reliable information as was available. He also ruled out conjecture and second-guessing. Comments would be based on known facts, even if there were gaps in the information coming from Soviet or other official sources.

During the next few weeks, both the facts and the radioactive clouds from Chernobyl spread slowly westward. EPA's Environmental Radiation Ambient Monitoring System (ERAMS)—continuously operated by ORP and augmented by reports from DOE national labs, the military, U.S. diplomatic missions abroad, and commercial nuclear power plants in this country—provided daily radiation measurements based on samples from hundreds of monitoring posts in the United States and abroad. The headquarters press office worked seven days a week until May 23, issuing daily task force reports and fielding thousands of in-person visits and phone calls from reporters all over the world. Chris Rice, press specialist for the radiation program, began to feel like the "voice of Chernobyl" as he handled phone calls from print and broadcast reporters.

"I Trained All My Life for This"

Five days after the Chernobyl explosion ORP/Las Vegas specialist Richard Hopper was home preparing for dinner when the telephone rang. By 11 that night he was on a red-eye flight to Washington. His luggage included a variety of hand-held radiation monitors and 60 "Thermal Luminescent Dosimeters," the familiar looking film badges we see in hospitals and laboratories. He was on his way to being EPA's man on the scene in Eastern Europe.

Hopper's mission? To monitor radiation levels in U.S. diplomatic missions in Poland, Hungary and Bulgaria, the Eastern European countries most directly in the path of meteorological systems carrying radioactive debris from the damaged Soviet reactor.

The next morning, the 43-year old, dark-haired Westerner met with other members of the team he was joining at EPA headquarters, then went to a briefing at the State Department, where the priority subject of discussion was whether to evacuate U.S. women and children in those countries. Hopper, whose regular job involves monitoring radioactivity and radiation exposure around the EPA Las Vegas Laboratories and the Nevada nuclear test site, suggested they hold the decision until he'd had a chance to check the actual radiation levels.

Arriving in Warsaw on May 3, he found the embassy people "full of apprehension. Anxiety definitely had taken over."

Many Poles were reluctant to accept their government's initial reassurances. In fact, he believes their concern helped the embassy attache speed him through Polish customs without having his instruments and equipment inspected.

En route from the airport, Hopper took readings in a number of places, including office buildings and houses, inside and outside. He found the readings to be very low. Because he had spent many years at the Nevada test site and had organized the monitoring network after the Three Mile Island incident, Hopper anticipated many questions he would be asked, but also assumed there would be problems in Europe that he hadn't heard about back home. The first day in Poland, he met with a team of Polish scientists (whose knowledge of the Las Vegas lab—one had actually been there—gave Hopper added credibility). They told him they were advising the populace to be wary of drinking milk and water, and eating vegetables that might contain radioactive particles.

That evening, he continued his monitoring activities until late at night. The next morning he spent two hours briefing the entire embassy staff, including families. He discussed the exposure levels he had found and the Polish scientists had recorded, putting the levels into a perspective that indicated there would be no long-term health effects for the embassy personnel and their families. After this, he met privately with individuals—mostly pregnant women—who had special concerns but were reluctant to discuss their fears in a public gathering. To Hopper, this was as important as talking to the larger group. He knew, from years of Nevada experience, that such concerns are "very real and frightening to the people involved. A danger you can't see or feel or smell can seem much worse than it really is."

Before leaving Poland, Hopper also went to Kracow and Poznan to monitor radiation levels and brief the U.S. consular staffs and their families; he also talked to the students and staffs at the schools attended by U.S. and British embassy children. He additionally managed a trip close to the border area closest to Chernobyl, where he took even more readings. His working days ran easily to 18 hours. Before leaving Poland, he set up a monitoring system at the embassy and trained the staff to use it over the next six months, a process he repeated in Hungary and Bulgaria.

In Hungary, too, where modern town laboratories were doing the monitoring, he found an openness about sharing information on the part of government authorities. And he found the same need for empathetic briefing of embassy people and their families. In Bulgaria, there was little sharing of information with him on the part of government officials, but his sessions with U.S. embassy personnel were comparable to those in Warsaw and Budapest.

Hopper has been on the EPA staff since the agency was founded, coming to EPA after serving at the Nevada test site and with the Public Health Service. He has three children, the oldest 24.

At the peak, eight press office staff members were dealing with media and other calls, although the pace slackened as it became apparent that the Chernobyl fire was out and that radiation levels in the United States were well below danger levels.

In addition to calls from the press, calls from organizations, congressional offices, and concerned individuals were also pouring into headquarters, the Regional Offices, and various EPA laboratories. At headquarters, responsibility for answering such inquiries was ultimately given to the Office of Public and Private Sector Liaison, which answered hundreds of individual calls and also circulated needed information through a system developed by the Centers for Disease Control to notify health officials of over-the-counter drug tampering incidents.

Most of the calls received by EPA offices concerned possible dangers to U.S. citizens traveling abroad, but others were concerned with reports of radioactivity in specific parts of the United States.

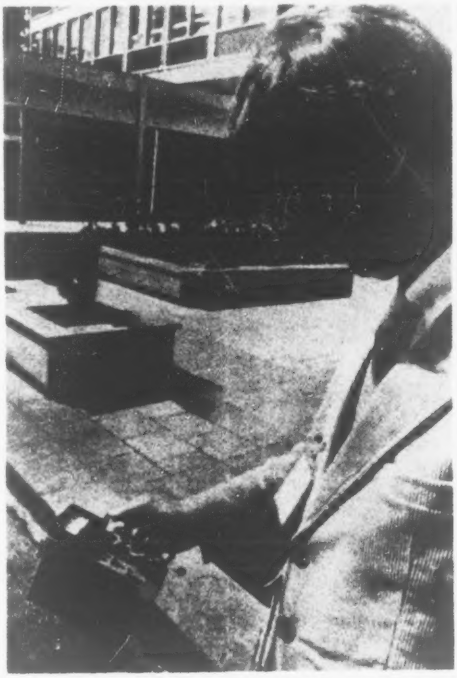
- Region 9 was called by a post office in Seattle which had received a parcel from Sweden. Was it safe to handle?
- Region 3 had a caller who wanted to know when the radioactive cloud would pass over Pittsburgh so she could take in her wash.
- Research Triangle Park had a call from a North Carolina dog breeder who had arranged to purchase an expensive German shepherd from a breeder in West Germany. He wanted to know if there had been heavy fallout in the

---

and his wife, Jacki, is a health physicist. Of this special assignment he says, "It was the opportunity of a lifetime. I've been training for this assignment for the last 20 years."

His feelings of satisfaction are more than echoed by a message from the U.S. Ambassador in Poland to the Secretary of State for relay to EPA:

"The entire staff of Embassy Warsaw joins me in expressing our heartfelt and most sincere thanks to Mr. Hopper for the outstanding manner in which he performed during his recent visit to Poland. His superb technical competence was perhaps expected, but he proved to be equally well qualified and adept at dealing with press inquiries, explaining his findings, reassuring worried mission members, and maintaining an invariably cooperative and cheerful attitude through long and very intensive workdays. He was highly professional in meetings with Polish scientific experts and obtained valuable information. He enthusiastically undertook three long and tiring field trips to various regions of Poland, making readings which enabled policy decisions to be taken in Washington. His serious but friendly manner and long experience made him particularly effective in visiting our diplomatic school and talking with pupils there. He . . . earned our unanimous admiration and respect." □

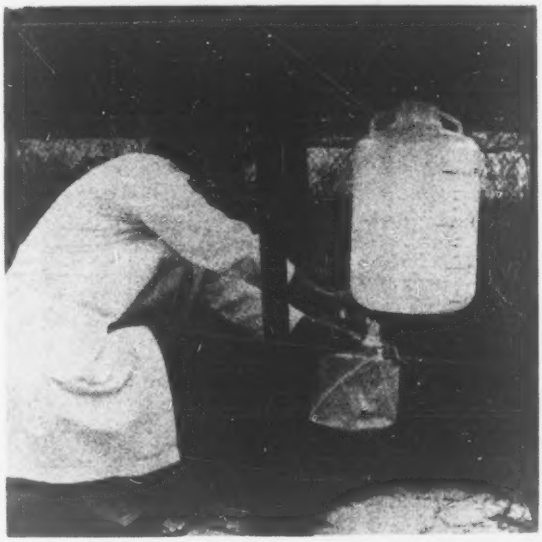


EPA radiation specialist Richard Hopper checks background radiation readings in front of the U.S. Embassy in Warsaw, Poland, on May 5, 1986.

dog's home so he could cancel the deal if the dog could get sick or harm his other dogs in North Carolina.

EPA's Office of International Activities (OIA) was also heavily involved. Radioactive debris from the reactor was monitored around the world and concern was high in most countries. OIA worked closely with the State Department to get radiation data on fallout within their borders. This information was used to inform the public of world-wide radiation levels and potential health risks for travelers. Richard Hopper of the ORP Las Vegas facility was sent to Poland, Hungary, and Bulgaria to work with U.S. Embassy officials monitoring potential impacts on U.S. employees there.

The Task Force met for the last time on May 14. Its last public report was issued on May 23, just short of four weeks after the accident. Reports from the Soviet Union are still adding new information. EPA's ERAMS system continues its regular monitoring activity, as does the instrumentation Hopper took to the U.S embassies abroad. The fallout from Chernobyl created no health problems for Americans here or abroad, but the nation now knows that, should another such accident occur or other radiation emergencies arise, there is an effective system in place to provide scientifically credible information about potential dangers and what to do about them. □



***At the Montgomery facility**, Avis Culver collects precipitation samples for radiation analysis. To monitor fallout from Chernobyl, EPA also provided daily radiation measurements based on air samples from hundreds of monitoring posts in the U.S. and abroad.*

Advice on Asbestos in the Home

by Dave Ryan

"Millions of Americans Face Asbestos Hazards in Homes" screams a headline in the *Atlanta Constitution*; "Is Asbestos Lurking in Your Home?" the *San Diego Union* asks ominously; "As Home Asbestos Crisis Grows Worse, Remedies Can be Costly and Dangerous," the *Wall Street Journal* warns in Armageddon-like tones.

True, home is where the heart is, but many Americans fear that it's also where the asbestos is—inside their domestic fortresses, creating time bombs of cancer in their very own kitchens and recreation rooms.

Approached with knowledge and caution, asbestos in any home can be safely brought under control.

It's easy to see how citizens can become apprehensive under such a barrage of headlines, but EPA has some advice: don't panic.

Asbestos is dangerous if inhaled, but its presence in your home is not necessarily a cause for alarm. As long as asbestos-containing material is maintained in good condition, there's usually no problem. It's only when this material is damaged that asbestos becomes dangerous, because then the fibers can escape into the air and be inhaled. (Soft, easily crumbled material, which EPA calls friable, has the greatest potential for releasing asbestos fibers into the air and therefore has the greatest potential to create health risks.)

In the majority of cases, then, these materials are best left in place. In fact, it's usually more dangerous to try to remove them; improper removal can disperse high levels of asbestos into the air.

Asbestos-containing materials can be found in many areas throughout the house, but these are the more common areas where asbestos may be found:

- **Pipe and Furnace Insulation**: Many homes built in the last 30 or 40 years have hot water and steam pipes and furnace ducts insulated with material containing asbestos. The most common type of this insulation contains asbestos mixed with paper, textile, or cement materials. These materials can often be repaired if the damage is minimal and in a confined area. Removal is recommended for damaged pipes when more than a small area has deteriorated.
- **Wall and Ceiling Insulation**: Homes built between 1930 and 1950 may have asbestos insulation sandwiched between exterior and interior walls. The quantities of asbestos involved may be substantial, and, correspondingly, so are the risks of exposing and disturbing it. Walls and ceilings should be checked for concealed asbestos insulation before beginning any repairs or renovations.
- **Stove and furnace insulation**: Asbestos has frequently been used to insulate wood-burning stoves as well as oil, coal, and wood furnaces. Usually the asbestos is contained in cement sheets, stiff paperboard, or paper. Some door gaskets in stoves, ovens, and furnaces may also contain asbestos.
- **Vinyl Floor Tiles and Vinyl Sheet Flooring**: Asbestos is often found in vinyl floor tiles and sheet flooring or their backing. If the flooring is sanded or cut to fit into place, or if old flooring is removed and the surface beneath it is sanded, fibers can be released into the air. To avoid disturbing asbestos fibers in existing flooring, it's best to place new flooring material directly over the old.
- **Patching Compounds and Textured Paint**: Homes built or renovated before 1975 may have patching compounds or textured paints with asbestos applied on wall or ceiling joints. If these materials are in good condition, it's best to leave them untouched. No patching compounds or textured paint should be sanded or scraped unless lab tests have confirmed them to be free of asbestos.
- **Walls and Ceilings**: In rare instances, private homes may have asbestos material sprayed or troweled on walls or ceilings, although this is much more likely to be found in large offices or apartment buildings built before 1973. Leave it undisturbed unless it shows signs of deterioration.
- **Roofing, Shingles, and Siding**: Asbestos-containing portland cement has sometimes been used to bind roofing and siding shingles. Since roofing and shingling are outdoors, intact materials pose minimal risk to building occupants; roofers who remove shingles, however, may experience a significant health threat unless they use proper procedures for handling asbestos-containing materials. Homeowners should avoid cutting, sanding, or scoring materials, although worn or damaged siding should be painted to seal in fibers that might otherwise escape.

What to do if you suspect there's asbestos in your home?

The first thing is to call EPA's TSCA (Toxic Substances Control Act) Assistance Office at the toll-free number (800) 424-9065 (554-1404 in the District of Columbia). They'll be glad to send a free packet including an asbestos fact sheet; a copy of a booklet called "Asbestos in the Home"; and a report titled "Guidance for Controlling Asbestos-Containing Materials in Buildings." The TSCA Assistance Office can also refer you to the Regional Asbestos Coordinator (RAC) in the nearest EPA regional office. The RAC offices are staffed by technical experts.

(Ryan is a Press Officer in the EPA Office of Public Affairs.)

architects, and engineers who have extensive practical experience with asbestos problems. In addition, many states now have licensing requirements for asbestos contractors. RACs can refer you to state contacts who can provide a list of licensed contractors.

For a thorough inspection of your living quarters, however, you'd better get some expert assistance.

Your local health department might inspect your home for free, or at least refer you to specially trained inspectors you can hire to do the job.

But, whoever does the work, you must remember that visual inspection alone is insufficient to identify asbestos. Laboratory analysis using polarized light microscopy is the only positive method of identification. Costs currently range from $20 to $50 per sample, and often several samples are required to confirm the presence of asbestos.

Samples must be collected in a way that protects the health of the sampler and ensures validity. The proper sampling procedures are spelled out in EPA's report, "Asbestos in Buildings: Simplified Sampling Scheme for Friable Surfacing Materials." If you're looking for a qualified laboratory near you, you can call another EPA toll-free number for help: (800) 334-8571, extension 6741.

If action must be taken to abate asbestos, it should only be done by a trained and qualified contractor. Home repair contractors are usually not experienced in the proper procedures for handling asbestos. A good abatement contractor will:

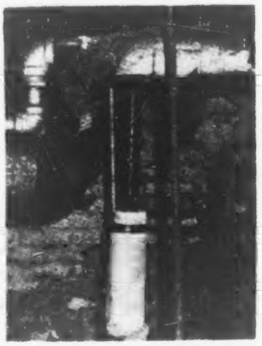
- Seal off the work area from the rest of the home with plastic sheeting and duct tape, and make sure not to track asbestos dust throughout the house;

- Always wear a respirator, protective gloves, and other protective clothing. Clothing should be disposed of as asbestos waste immediately after use. Clothing that cannot be thrown away should be washed separately from other clothes;

- Always wet asbestos-containing material before working with it. Wet asbestos fibers do not float in the air as readily as dry fibers, and are therefore easier to capture and dispose of;

- Avoid breaking asbestos-containing material into small pieces, which are more likely to release breathable fibers into the air;

- Place any debris from the work in 6-millimeter plastic trash bags and follow the instructions of the local health department for disposing of it in an approved landfill. The contractor should take care not to break the bag;



Environmental Management Systems, Inc.

Asbestos-coated hot water piping in a residential basement.

- Be sure not to dust or sweep particles suspected of containing asbestos, since this will disturb microscopic fibers and make them airborne. The fibers are so small that they cannot be seen. They can pass through normal vacuum cleaner filters and get back into the air. Dust should be removed by wet mopping and the use of High Efficiency Particulate Apparatus (HEPA) vacuum cleaners;

- Thoroughly clean the work area twice with wet mops, wet rags, or sponges, and be sure that no fibers are tracked into other areas of the home. All cleaning equipment should be disposed of in the same trash bags containing the asbestos materials.

It is important to find a contractor who conscientiously follows these safe work practices. In an effort to increase the numbers and availability of responsible contractors, EPA is encouraging all states to adopt certification programs ensuring that all asbestos abatement contractors are licensed to perform their work correctly.

Last year, EPA awarded grants to 12 states to support contractor certification programs, and this year plans awards to an additional 20 states. Partly as a result of this funding, 13 states now operate their own certification programs, and an additional 17 have certification bills either passed or pending in their legislatures. These states maintain lists of licensed contractors and make the lists available to the public.

Last year, EPA also opened three national training centers at Tufts University, Georgia Institute of Technology, and the University of Kansas. So far, these centers have instructed over 1,500 people, including contractors, building owners and managers, maintenance workers, school officials, architects, and consultants in various aspects of asbestos identification, maintenance, and control. Because of their success, EPA plans to open two more centers this year, one at the University of Illinois in Chicago and another at the University of California at Berkeley. In addition, four satellite centers will open late this spring to train contractors in proper abatement techniques. These will be located at Drexel University in Philadelphia, Rutgers Medical School in New Jersey, the University of Texas at Arlington, and the University of Utah in Salt Lake City.

In January of this year, EPA also proposed to ban five specific asbestos products for which there are adequate substitutes, and to phase out the use and importation of all other asbestos-containing products over the next 10 years. The ban will immediately prohibit the further sale and use in the U.S. of five products, the first three of which are used mainly in the construction and renovation industry: (1) saturated and unsaturated roofing felt; (2) flooring felt and asbestos felt-backed sheet flooring; (3) vinyl-asbestos floor tile; (4) asbestos-cement pipe and fittings; and (5) asbestos clothing. While EPA is not attempting to downplay the essential danger of inhaled asbestos, the Agency urges citizens not to let fear propel them into hasty and ill-conceived actions. When asbestos-containing materials are in good condition, the most appropriate action is to periodically inspect the materials for signs of damage or deterioration. If deterioration or damage is minor or localized, such simple steps as enclosing or repairing the material may be adequate. Only when asbestos-containing material is deteriorating over a broad area, or when it is likely to be disturbed by repair or renovation, should removal be considered, and then only by a trained contractor.

Approached with knowledge and caution, asbestos in any home can be safely brought under control. ☐

Update

A review of recent major EPA activities and developments in the pollution control program areas

AIR

Standards for Industrial Boilers

The Agency has proposed new rules under the Clean Air Act requiring reductions of sulfur dioxide emissions for new or modified industrial boilers.

The proposed rules require that new or modified steam generating units larger than 29 megawatts (100 million Btu/hour) achieve a 90 percent reduction in sulfur dioxide emission.

EPA rules are expected to apply primarily to industrial boilers but would include the largest institutional and commercial boilers and the smallest utility boilers.

The 90 percent reduction standard represents the performance capabilities of demonstrated control technologies over the 30 day averaging time of the standard.

ENFORCEMENT

Firm Indicted

A Pennsylvania firm and two of its officials have been indicted in connection with the storage and transportation of hazardous waste in south central Kentucky. The investigation was conducted by the EPA Office of Criminal Investigation in Region 4.

RAD Services Inc., Arthur J. Sciullo, Executive Vice President, and George R. Gary, head of the Chemicals Division, were indicted on five counts by a federal grand jury. According to U.S. Attorney Joseph Wittle, RAD illegally stored thousands of tons of hazardous waste in a Bowling Green warehouse, between 1980 and 1983, without notifying the EPA. The material was emission control dust, a byproduct of air pollution control devices in steel mills.

The investigation was prompted by complaints from residents of Rutherford County who became suspicious of late-night dumping in 1983.

HAZARDOUS WASTE

Superfund Priority List

EPA has added 170 hazardous waste sites to its final Superfund National Priorities List (NPL), making them eligible for long-term action under the Superfund site cleanup program.

The Agency also proposed 45 sites for the priority list. The proposed sites are subject to a 60-day public comment period and could be added at a later date.

With these additions, there are now 703 sites on the final NPL and 185 proposed sites. EPA deleted eight sites from the final NPL on March 7, 1986, since work on those sites had been completed.

PESTICIDES

Dicofol Registration Cancelled

EPA announced its decision to cancel the registration of the pesticide dicofol unless certain modifications are made to reduce significantly the levels of DDT and related contaminants in this product.

EPA is requiring two kinds of modifications to the dicofol registration. First, the levels of DDT allowed in dicofol will be reduced in two stages. There will be an initial and immediate reduction to less than 2.5 percent DDTr (DDT and related derivatives including DDD, DDE and, tetrachloro-DDT) contamination in the technical-grade compound. After December 31, 1988, all technical-grade dicofol products offered for sale must contain less than 0.1 percent DDTr. The second modification requires the registrants of dicofol to include a warning statement on labels of all products stating that loaders and applicators of the chemical should wear impervious gloves.

TOXICS

Information on Commercial Chemicals

The Agency has issued a final rule requiring chemical manufacturers and importers to report current production and plant-site information on thousands of commercial chemicals.

The new rule requires companies to report to EPA every four years beginning this year.

This rule requires the first substantial update of production and plant-site data for chemicals listed on EPA's chemical substances inventory. EPA will collect the latest information on chemicals in the current inventory on a plant-by-plant basis, including each chemical's identity, whether the firm is a manufacturer or importer, whether or not use of the substance is limited to the site where it is manufactured, how much is produced, the plant's technical contact, and whether any of the information is confidential.

WATER

Research Burn Permit Denied

EPA's Assistant Administrator for Water, Lawrence J. Jensen, has denied the application by Chemical Waste Management, Inc., for a research permit to conduct ocean incineration of toxic wastes.

Jensen said that his review of public comments and the hearing officer's recommendations had led him to conclude that it was not necessary to conduct the research burn at this time.

Chemical Waste Management had planned to conduct the research burn using the Vulcanus II, a special incinerator ship. The permit proposed burning 708,958 gallons of fuel oil containing 10 to 30 percent polychlorinated biphenyls (PCBs) at sea approximately 140 miles east of Cape May, NJ. ☐

Appointments/Awards

Thomas L. Adams, Jr.

Robie G. Russell

Lee DeHihns

Douglas D. Campt

Thomas L. Adams Jr. has been nominated to be Assistant Administrator for EPA's Office of Enforcement and Compliance Monitoring. Adams will be responsible for judicial enforcement actions against violators of federal environmental laws and for maintaining national consistency in the enforcement of the civil and criminal laws and regulations.

Since 1983 Adams served as the EPA Deputy General Counsel for Regional Coordination. From 1977 to 1983 he was assistant director for governmental relations for Republic Steel Corp. Prior to that he served for two years as assistant general counsel for the Federal Trade Commission and earlier as minority counsel for the Senate Commerce Committee's Subcommittee on Environment and Consumer Affairs.

Adams is a graduate of the University of Virginia and the Vanderbilt University School of Law.

Robie G. Russell has been named EPA's Regional Administrator for the Pacific Northwest Region (Region 10).

Russell has served as Senior Deputy Attorney General for the state of Idaho since 1981. As chief of the local government division, he has dealt with a broad range of issues including land use, elections, local government law, disaster planning, public meetings and records, and Indian law. He was Idaho Deputy Attorney General and acting chief of the natural resources division from 1979 to 1981.

Russell received a bachelor's degree in political science from the University of Idaho in 1973. He received a law degree from the University of Idaho in 1978.

Lee DeHihns, formerly the Associate General Counsel for Grants, Contracts and General Law, will become the new Deputy Regional Administrator in Region 4.

DeHihns, who has been with EPA since 1974, brings to this position an outstanding professional reputation. DeHihns has served at his present position since 1982. He previously served as an Attorney for the Office of the General Counsel, Acting Regional Counsel for Region 5, and Special Assistant to the Deputy Administrator.

DeHihns has received EPA's Bronze Medal and EPA's Special Achievement Award. He received his B.S. degree from the University of Scranton and his J.D. from the Columbus School of Law, Catholic University of America.

Douglas D. Campt has been appointed to be Director of EPA's Office of Pesticide Programs (OPP). He previously served as director of the Registration Division in OPP. Campt has been with EPA since 1970, serving as the Associate Director for Registration and Compliance, and as Program Management Officer. Prior to joining EPA, he was the head of the Registration Review Section, Assistant Chief of the Registrations Branch, and a Plant Quarantine Inspector at the Department of Agriculture.

Campt is a graduate of North Carolina Central University with a degree in biology. □

Performance Awards

Administrator Lee M. Thomas has presented awards to nine members of EPA management for "unusually outstanding" performance. These awards are made annually to those employees in the Performance Management and Recognition System whose job performance results in major progress toward Agency objectives.

The following persons received awards:

Gary A. Amendola, Supervisory Environmental Engineer, Environmental Services Division, Region 5.

Kathleen C. Callahan, Deputy Director, Office of Policy and Management, Region 2.

Thomas B. DeMoss, Director, Technical Support Division, Office of Marine and Estuarine Protection.

John A. Edwardson, Chief, Superfund RCRA Branch, Budget Division, Office of the Comptroller.

Chester J. France, Chief, Standards Development and Support Branch, Motor Vehicle Emissions Lab, Ann Arbor.

Denise M. Keehner, Chief, Regulatory Section, Exposure Evaluation Division, Office of Toxic Substances.

Walter E. Mugdan, Deputy Regional Counsel, Office of Regional Counsel, Region 2.

Gilman D. Veith, Associate Director for Research Operations, Environmental Research Laboratory, Duluth.

Thomas C. Voltaggio, Chief, Superfund Branch, Hazardous Waste Management Division, Region 3.

Kathleen Varaday inspects the air pump system attached to her home in Boyertown, PA. The system helps to ventilate radon from her home.

Back cover: Sailing. Photo by John Bowden, Folio, Inc.

RESCREEN & SQUARE HALFTONES

United States
Environmental Protection
Agency
Washington DC 20460

Official Business
Penalty for Private Use $300

Third Class Bulk
Postage and Fees Paid
EPA
Permit No. G-35

A. JUDSON WELLS
102 KILDONAN GLEN
WILMINGTON, DELAWARE 19807
PHONE 302-658-1791

SEP 23 REC'D

September 19, 1986

The Honorable James Scheuer
U. S. House of Representatives
Washington, D.C. 20515

Dear Mr. Scheuer,

It was a pleasure testifying before your Subcommittee on Natural Resources, Agriculture Research and the Environment last Wednesday. You and your committee members were very attentive to the testimony given and asked penetrating questions.

You asked one question in several parts, and I never really answered the last part. Specifically, you asked whether or not it was necessary to pursue these passive smoking questions to the point that we had exact answers as to how many deaths there were and exactly what the health effects might be. The answer to this part of your question is no. With a low level risk like passive smoking there will always be variations in the study results so the best we can do is strike some sort of an average, then apply a margin of safety and take whatever action is required. What makes passive smoking an important public health issue is that, in spite of the low level of risk, there are so many millions of people exposed that the potential number of deaths rises into the thousands.

For the case of female lung cancer, I would say that the case is pretty well proved. Yet even there my estimate of 600 deaths per year would be in a range of 325 to 875 if one took the 95% confidence limits from the combined epidemiological relative risk that I showed in my Table I (see enclosure). In every study on male lung cancer from passive smoking that I have seen the male relative risk is higher than the female relative risk, so even though we have many fewer cases for males, I think we can safely say that their risk is higher. The range for male deaths in my study would be 375 to 1775. Combining these male and female ranges one gets 700 to 2650 surrounding the figure of 1800 deaths a year that I quoted for lung cancer. Obviously, these are not exact numbers, but they are all large numbers, much larger than we normally worry about for a typical airborne carcinogen, and they agree well with Repace and Lowrey's range of 500 to 5000.

For heart disease and cancers other than lung some more work would be desirable. Here we have few results, and the potential adverse health effects are very large. It would be nice to know whether we are talking about 45,000 deaths per year as I suggested or 4500 or perhaps 75,000. Also the work might provide some clues as to how cigarette smoking causes these diseases.

Incidentally, I called Sam Arsht today to pass along your greetings. He was very pleased to hear of you and your activities in this field. He said you should report the bill favorably, whatever bill it was you had in mind. Also he said that you and his daughter appear in a photograph in this morning's New York Times, in case you haven't seen it. Apparently it was at some party that I believe she gave for a singer or some such. Small world!

As I told you at the meeting, if there is anything further that I can do to help on the passive smoking front I would be glad to oblige. And it was a pleasure making your acquaintance.

Sincerely,

A. Judson Wells

A. Judson Wells, Ph.D.

Incidentally, we appreciate very much help in getting our new Council on Clean Indoor Air up and running.

TABLES

for testimony of Dr. A. Judson Wells

September 17, 1986

TABLE I

COMBINED RISK RATIOS FOR PASSIVE SMOKING

Disease	Exposed Cases	R/R*	2-tail p	95% conf. int.
Female				
Lung cancer	463	1.44	<0.001	1.2 - 1.7
Other cancer	2091	1.56	<0.001	1.3 - 1.9
Ischemic heart dis.	276	1.27	0.04	1.0 - 1.6
Emphysema & chr. br.	102	1.4	0.18	0.9 - 2.1
Male				
Lung cancer	15	2.5	0.009	1.3 - 4.7
Other cancer	7	1.0	-	-
Ischemic heart dis.	14	1.3	0.46	0.7 - 2.6

range of relative risks around 1.44

range around 2.5

*Risk ratio or relative risk

TABLE II

PRELIMINARY RISK ASSESSMENT FOR PASSIVE SMOKING

PROBABLE NUMBER OF U.S. DEATHS PER YEAR

	Females			Males			Both
Non-smoking Population, 45+	27.6 MM			20.3 MM			47.9
% Exposed	76%			55%			67%
Passive smoking Population, 45+	21.0 MM			11.2 MM			32.2
Disease	R/R[a]	Non-smoker Death Rate[b]	Deaths from Pass. Smkg.	R/R[a]	Non-smoker Death Rate[b]	Deaths from Pass. Smkg.	Total Deaths
Lung Cancer	1.44	9	600	2.5	13	1,200	1,800
Other Cancer	1.56	245	20,200	1.0	188	0	20,200
Ischemic Heart	1.27	210	9,900	1.3	531	15,300	25,200
Emphysema	1.4	2	100	1.0	6	0	100
Total - four Diseases			30,800			16,500	47,300

range is 325 to 875 for 95% confidence interval

range is 375-1775

combining the ranges yields an overall range of 700 - 2650

[a] Relative risk.

[b] Per 100,000 person years.

October 1, 1986

The Honorable James H. Scheuer
Chairman, Natural Resources,
Agriculture Research and
Environment Subcommittee
Committee on Science and Technology
House of Representatives
Washington, D.C. 20515

Dear Mr. Chairman:

As a scientist who is familiar with the medical literature exploring the possible health consequences of human exposure to environmental tobacco smoke, I have been asked by The Tobacco Institute to address briefly the testimony that was presented to the Natural Resources, Agriculture Research and Environment Subcommittee by A. Judson Wells on September 17, 1986.

My experience and qualifications are set forth in the attached curriculum vitae. I also have included as part of this submission two presentations made earlier this year on the epidemiological literature cited by Wells. The first was presented to the National Research Council by Philip Witorsch, M.D., and is entitled "ETS and Cardiorespiratory Disorders." The second was prepared by Nancy Balter, Ph.D., et al. ("Causal Relationship Between ETS and Lung Cancer in Non-Smokers: A Critical Review of the Literature") and has been published in the proceedings of the 79th Annual Meeting of the Air Pollution Control Association. These papers discuss some of the limitations of the available health data on ETS and provide support for my more general comments.

Wells presented in his testimony the results of what he termed a "preliminary risk assessment for passive smoking." According to Wells, approximately 47,000 "preventable deaths" occur each year in the United States because of exposure to environmental tobacco smoke (ETS). Although Wells described that number, as well as the analysis upon which it was based, as "preliminary," he made no effort in his testimony to alert the members of the Subcommittee to the limitations of his analysis. Neither did he discuss, and perhaps was not aware of, the numerous assumptions that must be made before any risk analysis -- including a risk analysis for ETS -- is undertaken.

In my opinion, the "preliminary risk assessment" for ETS presented to the Subcommittee by Wells is misleading for several reasons. The first is that risk assessment computations have little meaning unless a causal relationship has been established between the substance being studied and a particular health effect. Despite Wells' assumption to the contrary, I do not believe that ETS has been shown scientifically to cause adverse health effects. Consequently, the most that Wells can say is that a substantial number of people would be at risk if ETS adversely affected human health. But that suggestion could be made about any number of ubiquitous substances that have not been shown to have any adverse health consequences. Second, Wells' description of the epidemiological investigations that form the basis of his risk calculations is both selective and erroneous. Third, Wells' risk calculations -- viewed exclusively from a statistical perspective -- are unclear and, in some instances, may be in error. And, finally, Wells has characterized his results in a manner that is likely to mislead anyone not familiar with risk assessment methods.

As a general proposition, quantitative risk assessment is a tool for estimating the risk to the general population associated with an activity or exposure that has been independently shown to cause a given effect. In the absence of an established causal relationship, quantitative risk assessment is largely meaningless. Thus, the first question that must be asked concerning the Wells presentation is whether ETS has been shown to cause adverse health effects among nonsmokers.

That question has been the subject of three important scientific conferences during the past few years. The conclusions reached at those conferences, as noted below, run flatly counter to Wells' causal assumption:

-- In May, 1983, a Workshop on Respiratory Effects of Involuntary Smoke Exposure was held in Bethesda, Maryland, under the auspices of the National Heart, Lung and Blood Institute, Department of Health and Human Services. The summary report of the workshop concluded that "the effect of passive smoking on the respiratory system . . . varies from negligible to quite small. From this review, it was not possible to determine whether there is a specific group which is at increased risk or what the mechanism of the effect (if any) may be."

-- In March, 1983, an international Workshop on Environmental Tobacco Smoke was held in Geneva, Switzerland, and its proceedings were published in the European Journal of Respiratory Diseases. The Workshop's organizer, Dr. Ragnar Rylander of the University of Gothenburg, Sweden, summarized the conclusions reached at the workshop as follows: "An overall evaluation based upon the available scientific data leads to the conclusion that an increased [lung cancer] risk for non-smokers from [environmental tobacco smoke] exposure has not been established."

-- In April, 1984, an international "Symposium on Passive Smoking from a Medical Point of View" was held in Vienna, Austria, organized by the American Health Foundation, and the Austrian, Bavarian and German Societies for Occupational Medicine. Following the symposium, Dr. Ernst Wynder, president of the American Health Foundation, summarized the participants' discussions as follows: "Should lawmakers wish to take legislative measures with regard to passive smoking, they will, for the present, not be able to base their efforts on a demonstrated hazard from passive smoking."

The scientific opinion reflected in the conferences described above is supported by the recently published findings of researchers from the Institute of Cancer Research in Great Britain. In summarizing their results, the British investigators noted: "Amongst lifelong non-smokers, passive smoking was not associated with any significant increase in the risk of lung cancer, chronic bronchitis, ischemic heart disease or stroke in any analysis." Lee, Chamberlain and Alderson, "Relationship of

Passive Smoking to Risk of Lung Cancer and Other Smoking-Associated Diseases," Br. J. Cancer 54:97-105 (1986).

Since ETS has not been shown scientifically to cause cancer or any other pathology, there is no logical basis for Wells' use of quantitative risk assessment to arrive at estimates of "avoidable deaths." This point is well illustrated by a similar misuse of risk assessment methodology by Repace and Lowery, to which Wells referred in both his testimony and paper. The Office of Technology Assessment (OTA) of the United States Congress rejected Repace and Lowery's conclusion that ETS annually causes between 500 and 5,000 deaths from lung cancer. In so doing, OTA criticized the authors for, among other things, assuming:

> "[T]hat the entire difference between the lung cancer death rate in a group of nonsmoking Seventh Day Adventists and in a group of nonsmoking (non-Seventh Day Adventists) Southern Californians was attributable to passive smoking. Mortality rates for cancers at other sites are also lower in that Seventh Day Adventist population, and the exact reasons for the differences are not all known. . . . At best, one can conclude that some part of the difference between the two populations may be due to difference in passive smoking rates, but the assumption that it is reasonable to attribute the entire difference to passive smoking is unjustified. The effect of these and other flaws on the final estimates calls into question the reliability of either of these numbers."

Office of Technology Assessment, U.S. Congress, Staff Paper, Passive Smoking in the Workplace: Selected Issues 21 (May 1986). The papers by Drs. Witorsch and Balter, *et al.*, that are attached discuss the pertinent epidemiological investigations in detail.

Even if a quantitative risk assessment with respect to ETS could be said to be appropriate, Wells' mortality figures would be of doubtful value because of uncertainty over the methodology used by him in applying risk assessment theory. Wells dismisses this concern in his paper by asserting that "it is not possible in a paper of this scope to give a detailed and sophisticated explanation of each step." But such explanation is critical to any meaningful evaluation of the conclusions Wells presented to the Subcommittee.

From the materials that are available, it appears that Wells' analysis involves three levels of suspect mathematical manipulation. First, Wells combined data from individual studies across exposure groups to yield a single value for relative risk of "exposed" versus "nonexposed" subjects. In many cases, the values presented by Wells are inconsistent with the data reported in the original study. Second, Wells combined "relative risks" from several selected studies to yield a single composite value for each cause of death and sex. Because the method used to combine individual relative risks was reported in a doctoral thesis, and is not therefore available in the peer reviewed literature, it is impossible to determine whether the method was applied appropriately. Similarly, the last manipulation, the calculation of annual deaths based on the composite relative risk values, has been incompletely described and therefore cannot be evaluated.

It is important to understand, in addition, that any conclusions derived by whatever method from underlying data can be no more reliable than the data itself. Wells' conclusions are flawed, in further part, because the scientific literature that Wells relied upon is inferior both in quality and quantity. In his paper, Wells characterized his risk assessment for female lung cancer as "the most reliable" of his numbers, and he also used that disease to illustrate his testimony. The few papers upon which Wells relied to support his "strongest" case cannot carry the load he places upon them.

As more fully explained in the accompanying paper by Dr. Balter (for which I was a coauthor), the literature on ETS exposure and lung cancer in nonsmoking females is characterized by inconsistencies both within and among studies. In addition, most of the studies suffer from critical methodological weaknesses -- a point that Wells appears to acknowledge. The most pervasive problem in the studies is the failure adequately to document exposure to ETS. Without that documentation, there is no way to tell whether a particular health effect was caused by ETS or some other substance or factor.

The majority of epidemiologic studies have compared lung cancer deaths in nonsmoking women married to smokers to the incidence of lung cancer deaths in such women married to nonsmokers. In most studies, information on exposure was based on personal interviews or a questionnaire that asked about the smoking status of the patient and her spouse. In some cases, an attempt was made to quantitate exposure by asking how many cigarettes a day the spouse smoked or for how long he had been smoking. An independent study (Friedman) has demonstrated that this approach is not valid for estimating exposure to ETS. In that study, 47 percent of the nonsmoking wives of smoking husbands reported no exposure to ETS in the home. At the same time, forty percent of nonsmoking women married to nonsmokers reported significant ETS exposure outside the home. Therefore, studies that estimate the risk of lung cancer as a function of smoking by the spouse are not necessarily measuring lung cancer risk as a function of ETS exposure.

The significance of this distinction is illustrated by the recent study by Lawrence Garfinkel of the American Cancer Society. Great care was taken in the Garfinkel study to document ETS exposure by a combination of questionnaires and interviews. Information on ETS exposure included not only the number of cigarettes smoked by the husband per day but also the number of cigarettes smoked by the husband at home, exposure to ETS in the workplace, and elsewhere. Garfinkel reported an increased incidence of lung cancer in the female spouse with increasing numbers of cigarettes smoked by the husband at home. But he also found no statistically significant relationship between the total hours per day of ETS exposure (at home, at work and elsewhere) and the risk of lung cancer. These findings suggest that spousal smoking is a very poor surrogate for ETS exposure. They also tend to suggest that any relationship between ETS and lung cancer is due to factors other than ETS.

In addition to these fundamental problems of approach and methodology, Wells' presentation suffers from a variety of technical flaws that would be readily apparent to anyone acquainted with the epidemiology of ETS and risk assessment. A couple of significant examples should serve to illustrate those problems.

Wells sets forth in Table II of his testimony the number of excess deaths he claims are caused each year in men because of exposure to ETS. That includes 15,300 excess deaths from ischemic heart disease and 1,200 excess deaths from three other diseases that he considers. Wells reports in Table I, however, that the p value (which he defines incorrectly in his testimony) is 0.46 for male ischemic heart disease, indicating that the risk that he reports in Table II is not statistically significant. What that means to a non-statistician is that, even using Wells' criteria, no excess deaths in men from ischemic heart disease can be attributed to ETS. Indeed, given the confidence limits that Wells has reported, it could as persuasively be argued that as many as 17,842 male deaths per annum were avoided as a result of ETS exposure. Obviously, neither of those conclusions would be appropriate.

Similarly, Wells' combination of studies employing different methods (prospective and retrospective) suggests that he has had little or no training in basic epidemiologic methods. He does not appear to be aware of the dangers of combining what are, in effect, apples and oranges from an epidemiological perspective. The figures produced by such combination represent nothing more than a statistical artifact that confuse, rather than illuminate, the issues that Wells addresses.

In sum, the Wells presentation must be viewed with extreme caution. I doubt very much that any reputable scientist would take Wells' presentation seriously, and I would hope that members of the Subcommittee are not misled by the presentation.

I would appreciate your adding this letter and its attachments to the record of the Subcommittee's September 17 hearing.

Sincerely,

SJ Kilpatrick Jr.
S. James Kilpatrick, Jr.
Medical College of Virginia

2/84

CURRICULUM VITAE

S. James Kilpatrick, Jr., Ph.D.

Date and place of birth: April 24, 1931; Belfast, Northern Ireland
U.S. Citizen

Social security number: 229-68-3208

Home address and telephone number:
7808 Ardendale Road
Richmond, VA 23225
(804) 272-8160

Office address and telephone number:
Department of Biostatistics
Medical College of Virginia
Virginia Commonwealth University
Box 460, MCV Station
Richmond, VA 23298
(804) 786-9824

EDUCATION

Ph.D., Medical Statistics, The Queen's University of Belfast, Belfast, Northern Ireland, 1960

M.Sc., Genetic Statistics, The Queen's University of Belfast, 1957

B.Sc., Pure and Applied Mathematics, The Queen's University of Belfast, 1954

POSTDOCTORAL TRAINING

1973-1974	Honorary Research Fellow Royal College of General Practitioners Research Unit Birmingham, England
1960-1961	U.S. Public Health Service Postdoctoral Fellow Department of Statistics, Iowa State University Ames, Iowa

ACADEMIC APPOINTMENTS

1965-present	Professor Department of Biostatistics, Medical College of Virginia Richmond, Virginia
1965-1983	Chairman Department of Biostatistics, Medical College of Virginia Richmond, Virginia
1979-1983	Professor Department of Family Practice, Medical College of Virginia Virginia Commonwealth University, Richmond, Virginia

ACADEMIC APPOINTMENTS (continued)

1961-1965	Lecturer Joint appointment: Department of Social Medicine/Department of Statistics, University of Aberdeen, Aberdeen, Scotland
1958-1961	Lecturer in Medical Statistics Department of Social and Preventive Medicine, The Queen's University of Belfast, Belfast, Northern Ireland
1954-1958	Assistant Lecturer in Medical Statistics Department of Social and Preventive Medicine, The Queen's University of Belfast, Belfast, Northern Ireland

MEMBERSHIP - SCIENTIFIC, HONORARY and PROFESSIONAL SOCIETIES

International Epidemiological Association, 1978-present
World Organization of National Colleges, Academies and Academic Associations of General Practitioners/Family Physicians, 1976-present
North American Primary Care Research Group, 1975-present
Sigma Xi, 1970-present
Virginia Academy of Sciences, 1965-present
American Statistical Association, 1961-present
Biometric Society, 1961-present
The American Public Health Association, 1974-1980
Society for Epidemiological Research, 1969-1980
Fellow, Royal Statistical Society, 1957-present

SPECIAL AWARDS, FELLOWSHIPS and OTHER HONORS

Personalities of the South
Who's Who in America
American Men of Science
Who's Who in Technology Today

MAJOR COMMITTEES

Ad hoc Reviewer, National Institute of Mental Health, National Cancer Institute
Special Review Committee for NHLBI RFA Studies of Cardiac and Vascular Disease in relation to Diabetes Mellitus (1977)
Chairman, The Rockefeller Foundation Workshop on the Denominator Problem in Family Practice, Bellagio, Italy, September 1981
Parent Review Committee, NHLBI-DBDR-816-A, Comprehensive Sickle Cell Center, 1981
Clinical Trials Review Committee; for NHLBI RFP 81-3 "Community Surveillance of Cardiovascular Diseases", 1981; for NHLBI RFP 82-07 "Cardiac Arrhythmia Pilot" Co-ordinating Center; for NHLBI RFP 82-12 "Thrombolysis in Myocardial Infraction (TIMI)" Co-ordinating Center
Member, MCV/VCU Cancer Center, 1980-present
Chairman, The Rockefeller Foundation Workshop on the Denominator Problem in Family Practice, New York City, September 1979

MAJOR COMMITTEES (continued)

Member, Oral Biology and Medicine Study Section, National Institutes of Health, 1978-1980
Member, Virginia Health Interview Council, 1977-1980
Member, Regional Advisory Board, Eastern North American Region, Biometric Society, 1977-1979
Member, Virginia Health Statistics Advisory Council, 1976-present (Chairman, 1979-present)
Consultant, Ambulatory Care Review Demonstration Project, South Central Pennsylvania PSRP (Area IX), 1976-1978
Member, Subcommittee on Biometry, Southern Regional Education Board, 1971-1978 (Chairman, 1976-1978)
Member, Committee on Statistics, Southern Regional Education Board, 1970-1978
Chairman, Virginia Chapter, American Statistical Association, 1966-1967
Chairman, Statistics Section, Virginia Academy of Sciences, 1966-1967

OTHER SIGNIFICANT SCHOLARLY or RESEARCH EXPERIENCE

Co-Investigator
National Institute of Dental Research #DE 05054
"Microbial Studies of Periodontal Disease"
$256,759 December 1, 1981 - November 30, 1986

Co-Principal Investigator
National Cancer Institute #5 P30 CA 16059
"MCV-VCU Cancer Center Core Grant - Epidemiology/Statistics Unit"
$537,854 January 1, 1982 - January 1, 1985

Co-Investigator
National Institutes of Health #5 P50 DE05139
"Clinical Research Center for Periodontal Disease"
$2,168,878 October 1, 1978 - September 30, 1983

Principal Investigator
The Rockefeller Foundation Grant-In-Aid #GA HS 8013
"Estimation of the Population Served by a Family Practice"
$75,000 May 1, 1979 - April 31, 1982

Co-Principal Investigator
Family Residency Training Grant #03D005910-02
"Curriculum Audit and Care by Practice Measurement"
$141,000 July 1, 1976 - June 30, 1977

Principal Investigator
National Center for Health Services Research Grant #1 R01 HS 01899
"Evaluation of the Demand for Primary Care in Virginia"
$233,000 July 1, 1975 - June 30, 1978

Principal Investigator
National Institute of Mental Health Grant #1 R03 MH 24717
"Coding and Disposition of Psychosocial Illness"
$5,800 June 1, 1974 - May 31, 1978

OTHER SIGNIFICANT SCHOLARLY or RESEARCH EXPERIENCE (continued)

Principal Investigator
Division of Research Resources, Biotechnology Branch, NIH Grant #5 PO 7 RR00016
"Expansion of Scientific Computation Laboratory"
$444,000 October 1, 1967 - August 31, 1973

Director
Departmental Statistical Laboratory, 1979-1981

Organizer and Chairman
The Rockefeller Foundation supported conference
"Research in Primary Care: Encounter Records and the Denominator"
Villa Serbelloni, Bellagio, Italy September 28 - October 2, 1981

Director
Departmental Biostatistics Information System, 1979-present

Coordinating Statistician
Multi-center clinical trial of chlorpropamide, British Diabetic Association, 1963-1964

Referee
Journal of the American Medical Association, Journal of Family Practice, Biometrics

BIBLIOGRAPHY

Attached as separate document.

2/84

BIBLIOGRAPHY

S. James Kilpatrick, Jr., Ph.D.

JOURNAL PUBLICATIONS

1. Kilpatrick, S.J., Mathers, J.D., and Stevenson, A.C.: The importance of population fertility and consanguinity data being available in medico-social studies. The Ulster Medical Journal, 24: 113-122, 1955.
2. Cheeseman, E.A., Kilpatrick, S.J., Stevenson, A.C., and Smith, C.A.: The sex ratio of mutation rates of sex-linked recessive genes in man with particular reference to Duchenne type muscular dystrophy. Annals of Human Genetics, 22(3): 235-243, 1958.
3. Smith, C.A.B. and Kilpatrick, S.J.: Estimates of the sex ratio of mutation rates in sex-linked conditions by the method of maximum likelihood. Annals of Human Genetics, 22(3): 244-249, 1958.
4. Findlay, I.A. and Kilpatrick, S.J.: Myographic aspects of the swallow. Journal of Dental Research, 38: 1219, 1959.
5. Findlay, I.A. and Kilpatrick, S.J.: An analysis of the sounds produced by the mandibular joint. Journal of Dental Research, 39(6): 1163, 1960.
6. Murphy, F.M., Barber, J.M., and Kilpatrick, S.J.: Tersavid in angina pectoris. A sequential clinical trial. The Lancet, 139-140, 1961.
7. Kilpatrick, S.J.: Occupational mortality indices. Population Studies, 16: 175, 1962.
8. Kilpatrick, S.J.: Mortality comparisons in socio-economic groups. Applied Statistics, 12(2): 64, 1963.
9. Imray, J.M., Kennedy, B.R., and Kilpatrick, S.J.: The effect of brief chloroform administration on liver function. Anaesthesia, 19(1): 33, (1964).
10. Richardson, I.M. and Kilpatrick, S.J.: Influenza immunization, a three year study in a factory population. The Medical Officer, 111: 5-7, 1964.
11. McKay, E. and Kilpatrick, S.J.: Maternal and infant plasma urea at delivery. The Journal of Obstetrics and Gynecology of the British Commonwealth, 71: 449, 1964.
12. Kilpatrick, S.J. and McKay, E.: Umbilical cord urea in toxemia of pregnancy. The Journal of Obstetrics and Gynecology of the British Commonwealth, 72: 402, 1965.
13. Blair, D.W., Williams, M.J., Carr, A.J., and Kilpatrick, S.J.: Effect of L-thyroxine on gastric secretion in the pylorus-ligated rat. The Journal of British Society of Gastroenterology, 6: 343, 1965.
14. Caridis, D.T., Blair, D.W., Kilpatrick, S.J., and Carr, A.J.: The Effect of alloxan on gastric secretion in pylorus-ligated rats. Scottish Medical Journal, 11: 247, 1966.
15. Hume, D.M., Williams, G.M., White, H.J.O., Ferre, J., Wolf, J.S., Prout, C.R., Slapek, M., O'Brien, J., Kilpatrick, S.J., Kauffman, H.M., and Cleveland, R.J.: Comparative results of cadaver and related donor renal homografts in man and immunologic implications of the outcome of second and paired transplants. Annals of Surgery, 164: 352, 1966.

JOURNAL PUBLICATIONS (continued)

16. Kilpatrick, S.J. and Gamble, E.: A model for incompatibility in kidney transplants. Annals of Human Genetics, 31: 309, 1968.
17. Kilpatrick, S.J.: Graphical estimation of the number of strong histocompatibility antigens present in renal homografts. Annals of Human Genetics, 32: 305, 1969.
18. Leo, J.R., Kilpatrick, S.J., DePlanque, B.A., Williams, G.M., and Hume, D.M. A genetic model of human histocompatibility based on the results of double cadaver renal homografts. Transplantation Proceedings, 1: 379, 1969.
19. Kilpatrick, S.J.: An alternative to the standardized score in grading a multiple choice examination. Journal of Experimental Education, 39(4): 61, 1971.
20. Kilpatrick, S.J.: Collusion in multiple choice examinations. MCV/Quarterly, 7(1): 3, 1971.
21. Kilpatrick, S.J.: Alternative methods of grading one or more multiple choice examinations. MCV Quarterly, 7(1): 4-9, 1971.
22. Wise, R.M., Rondinone, J.F., and Briggs, F.N. (with an Appendix by S.J. Kilpatrick and W. Stuart): Effect of pCa on series-elastic component of glycerinated skeletal muscle. American Journal of Physiology, 224(3): 576-579, 1973.
23. Kilpatrick, S.J.: The distribution of episodes of illness - a research tool in general practice? Journal of the Royal College of General Practitioners, 25: 686-690, 1975.
24. Kilpatrick, S.J.: Factors in morbidity: Inter-practice variation of episodes illness reported in 1970-71 national morbidity survey. Journal of the Royal Statistical Society-C, 26(1): 26-33, 1975.
25. Kilpatrick, S.J.: Consultation frequencies in general practice. Health Services Research, 12(3): 284-298, 1977.
26. Kilpatrick, S.J. and Wood, M.: A comparison of the morbidity record in two family practice surveys. The Journal of Family Practice, 4(5): 972-973, 1977.
27. Kilpatrick, S.J.: Successes and failures in the analysis of family medicine data bases. Proceedings of the 18th National Meeting Public Health Conference on Records and Statistics, Capitol Hilton, Washington, D. C., August 4. New Challenges for Vital and Health Records, 35-38, 1980.
28. Kilpatrick, S.J. and Krogh-Jensen, P.: The denominator problem. Journal of Family Practice, 10(2): 344, 1980.
29. Rockhold, F.W. and Kilpatrick, S.J.: Methods of discrimination among stochastic models of the negative binomial distribution with an application to medical statistics. Biometrische Zeitchrift, 23(7): 681-692, 1981.

ABSTRACTS

1. Findlay, I.A. and Kilpatrick, S.J.: Analysis of myographic records of swallowing in normal and abnormal subjects. Journal of Dental Research, *39*: 629, 1960.
2. Kilpatrick, S.J.: A model for incompatibility in kidney transplants. Biometrics, *24*: 212, 1968.

BOOKS AND/OR CHAPTERS

1. Backett, E.M., Sumner, G., Kilpatrick, S. J., and Dingwall-Fordyce, I.: "Hospitals in the north-east Scotland region. Problems and progress in medical care assays on current research," Studies of Hospital Patient Services, 2nd series, G. McLachlan, ed., London: Oxford University Press, 1966.
2. Kilpatrick, S. J.: *Statistical Principles in Health Care Information*, 1st edition, 1973 (2nd edition, 1977). Baltimore: University Park Press.
3. Kilpatrick, S.J. and Boyle, R.M., (eds.): *Primary Care Research*: *Encounter Records and the Denominator*. New York: Praeger 1984.
4. Kilpatrick, S.J.: An evaluation of family practice denominators. Chapter 3, pp. 41-49. In Kilpatrick, S.J. and Boyle, R.M., (eds.), *Primary Care Research*: *Encounter Records and the Denominator*. New York: Praeger 1984.
5. Kilpatrick, S.J.: Indirect estimation of non-attending patients using the negative binomial. Chapter 4, pp. 51-64. In Kilpatrick, S.J. and Boyle, R.M., (eds.), *Primary Care Research*: *Encounter Records and the Denominator*. New York: Praeger 1984.
6. Rockhold, F.W. and Kilpatrick, S.J.: Estimation of the populations at risk in general practice using truncated information from the negative binomial. Chapter 5, pp 65-81. In Kilpatrick, S.J. and Boyle, R.M., (eds.), *Primary Care Research*: *Encounter Records and the Denominator*. New York: Praeger 1984.
7. Kilpatrick, S.J. and Boyle, R.M.: Summary and Recommendations. Chapter 9, pp. 145-149. In Kilpatrick, S.J. and Boyle, R.M., (eds.), *Primary Care Research*: *Encounter Records and the Denominator*. New York: Praeger 1984.
8. Kilpatrick, S.J.: A solution to the denominator problem? Chapter 10, pp. 151-154. In Kilpatrick, S.J. and Boyle, R.M., (eds.), *Primary Care Research*: *Encounter Records and the Denominator*. New York: Praeger 1984.

OTHER

Letters

1. Kilpatrick, S.J.: Mathematical models of general practice. Journal of the Royal College of General Practitioners, *26*: 454, 1976.
2. Kilpatrick, S.J.: Consultation. Journal of the Royal College of General Practitioners, *31*(226): 314-315, May 1981.
3. Kilpatrick, S.J.: Lymphadenopathy incidence. Journal of Family Practice, *13*(2): 164, August 1981.

Reviews

1. Kilpatrick, S.J.: Some malpractices in medical statistics. MCV/Quarterly 2(3): 207-211, 1966.

2. Kilpatrick, S.J.: Computers in the service of medicine, G. McLachlan and R.A. Shegoy, eds. London: Oxford University Press, 1968.

3. Kilpatrick, S. J.: The role of operations research in a university hospital, a review and bibliography. MCV/Quarterly 5(2): 61, 1969.

4. Kilpatrick, S.J. and Wood, M.: Analysis and interpretation of data. Special issue on research in family practice. Journal of Family Practice, 7(1): 101-112, 1978.

5. Anderson, J.E., Bass, M., Boyle, R.M., Freeman, D.M., Kilpatrick, S.J., Wood, M., and Froom J.: A glossary for primary care. Report of the North American Primary Care Research Group (NAPCRG) Committee on Standard Terminology. Journal of Family Practice, 5(4): 635-638, 1977.

6. Kilpatrick, S.J.: Mainland's elementary medical statistics, reprint of 2nd edition. Biometrics, 35(4): 902, 1979.

ETS AND CARDIORESPIRATORY DISORDERS

Philip Witorsch, M.D.
Clinical Professor of Medicine
Division of Pulmonary Diseases and Allergy
Department of Medicine
The George Washington University Medical Center
Washington, D.C. 20037

National Academy of Sciences Presentation

ETS and Cardiorespiratory Disorders

Philip Witorsch, M.D.
Division of Pulmonary Diseases and Allergy
Department of Medicine
The George Washington University Medical Center

This presentation focuses on the possible effects of Environmental Tobacco Smoke ("ETS") on the cardiovascular and respiratory systems in adults and children. Before examining the individual studies that have attempted to address this area, some preliminary observations may be in order.

Several criteria must be satisfied, of course, before a study can be said to support a causal hypothesis that would link environmental exposure -- whether to ETS or any other substance or agent -- and adverse health effects. The following criteria apply to both epidemiological and clinical experimental studies:

1. Estimates or measurements of exposure and/or dose must be accurate and verifiable. This means, among other things, that any questionnaires or instruments that are used must be appropriate to the situation, standardized, validated and used in a scientifically acceptable manner.

2. The study must be rigorously and appropriately designed. This requires adequate sample size, the use of appropriate study and control groups, including acceptable

environmental or exposure controls where pertinent, elimination or minimization of selection and observer bias and appropriate attention to potential co- and confounding variables. So far as ETS is concerned, the potential co- and confounding variables include, among other things, lifestyle, diet and nutrition, occupational and avocational exposure to substances other than ETS (such as fungi, bacteria, other microorganisms, ozone, radon and radon progeny, formaldehyde, asbestos and other respirable dusts, NO2, SO2, allergens, insect and rodent products, plants and plant products, pesticides, herbicides and household chemicals), socioeconomic factors, health attitudes and habits (including current and prior active smoking on the part of "exposed" subjects), genetic factors, preexisting diseases and conditions (including atopy), levels of ventilation and relative humidity, and psychogenic and emotional factors (including the influence of publicity).

3.. In interpreting a particular study, assuming it has been well designed and executed, differences in results between groups must, of course, be statistically significant, using generally accepted evaluative techniques and criteria.

4. Results must be internally consistent and, where applicable, consistent with data from other acceptable studies (*i.e.*, externally consistent).

5. Where the results imply an effect, a dose-response relationship should be demonstrable.

6. In addition to being statistically significant, any differences that are noted should be physiologically and clinically significant, biologically and clinically relevant and biologically plausible.

7. Whenever possible, both subjects and investigators should be "blinded" in an effective and appropriate manner.

8. Finally, the results should be reproducible by other investigators.

Respiratory System -- Adults

It has been suggested that data in the literature demonstrate that exposure to ETS can cause or exacerbate chronic obstructive pulmonary disease in adults, lead to small airways disease and dysfunction, initiate or exacerbate asthma, and adversely affect respiratory function and symptomatology in individuals with preexisting respiratory disorders. In fact, a careful review of the pertinent studies reveals that such suggestions can be accepted only if one is willing to ignore accepted scientific criteria and evaluation techniques.

Perhaps the most serious, and certainly the most widespread, flaw in the epidemiologic studies addressing ETS and adult pulmonary function is the absence of appropriately verified estimates or measurements of exposure and/or dose. It is therefore difficult, if not impossible, to reach scientifically reliable and meaningful conclusions regarding any cause-and-effect relationships. In addition, many of the studies, both epidemiological and experimental, suffer from other serious flaws in design and methodology that invalidate any cause-and-effect conclusions.

I hasten to point out that the invalidating flaws to which I refer frequently apply across the board -- that is, to studies purporting to show an adverse effect of ETS on adult pulmonary function as well as to those not demonstrating such an effect. One cannot overemphasize the fact that significantly flawed studies do not permit one to reach valid scientific conclusions and can neither prove nor disprove a hypothesis. Similarly, uncontrolled anecdotal observations, while a suitable basis for the initiation of studies and hypotheses, are by themselves not a valid basis upon which to reach scientific conclusions.

While it may seem intuitively reasonable to postulate a possible adverse effect of environmental tobacco smoke on respiratory function based upon primary or active smoking studies, any such analogy would be misleading. There are significant quantitative as well as possible qualitative differences between ETS and the mainstream smoke to which the active smoker is exposed (Brain and Barry, 1985; First, 1985), and the differences are likely to be significant so far as effects on the respiratory tract and pulmonary function are concerned. Consequently, the conclusion that ETS adversely affects lung function in the nonsmoker can be based only upon properly designed and conducted scientific studies focusing on ETS.

If one wishes to evaluate the effects of ETS exposure on pulmonary function, it is necessary that the pulmonary function testing instrumentation, methodology and analysis be appropriate and acceptable. This requires reliable, validated equipment that is properly maintained and calibrated; the performance of an adequate number of maneuvers by each subject; and strict adherence to acceptable criteria of reproducibility and for selection of sets of data for analysis (American Thoracic Society, 1979; Morris, et al., 1984). It is essential that the technician performing the testing be appropriately trained and experienced, blinded to the exposure status of the subject, and keenly aware of the significant effects of subject effort on the results obtained. In analyzing and interpreting the data, it is critical that the values obtained be compared with accepted predicted values for each subject based on age, body size, sex and race and that criteria of abnormality or deviation take into account the significant intrasubject variability that exists for many pulmonary function parameters. Additionally, the individual interpreting the results should be appropriately trained and experienced, as well as blinded to the exposure status of the subjects whose data are being evaluated (Lebowitz, et al., 1982a; Burrows, et al., 1983; Snow, 1982; Suratt, et al., 1981).

It is, furthermore, of paramount importance that the parameters selected for study indeed validly reflect the physiological function being evaluated. Indices derived from the forced expiratory spirogram, especially the FEV_1, are highly validated and useful for the assessment of airflow limitation, although they may leave something to be desired in terms of sensitivity and specificity (Miller, Grove & Pincock, 1985; Miller, Pincock & Grove, 1985; Fletcher, et al., 1976; Buist and Ducic, 1979; Bates, 1973; Johnston, et al., 1976). On the other hand, the physiological and clinical significance of the various tests that have been proposed to assess small or peripheral airways function is open to question and remains a subject of current controversy (Burrows, et al., 1983; Lebowitz, et al., 1982a; McFadden & Ingram, 1980; Berend & Thurlbeck, 1982; Meadows, et al., 1980; Mink & Wood, 1980; Beus, et al., 1981; Nagai, et al., 1985; Cosio, et al., 1977; Miller, Grove & Pincock, 1985; Miller, Pincock & Grove, 1985; Nemery, et al., 1981; Tattersall, et al., 1978). The purported "gold-standard" for peripheral airways dysfunction, frequency dependence of dynamic compliance, is cumbersome, technically difficult, requires considerable subject cooperation, and is thus not practical for use in epidemiologic or other studies of large groups of subjects (Burrows, et al., 1983). Mid- and terminal flow rates are associated with great within-subject and between-subject variability and are of uncertain physiological and clinical relevance, especially in the presence of a normal FEV_1 and FEV_1/FVC ratio (Burrows, et al., 1983; Beus, et al., 1981; Lebowitz, et al., 1982a; Miller, Pincock & Grove, 1985). Recent studies indicate that tests of density dependence of airflow which compare expiratory flow breathing helium-O_2 mixtures with that breathing air are of extremely doubtful validity as indices of small airway disease (Berend & Thurlbeck, 1982; Meadows, et al., 1980; Mink & Wood, 1980). Closing volume, closing capacity and the slope of the phase III alveolar plateau during the single-breath nitrogen test likewise have uncertain physiological and clinical meaning (Burrows, et al., 1983).

At the present time, therefore, it is extremely difficult to validly and meaningfully assess small or peripheral airways function in epidemiologic or other large group studies. But even should this become practical, the results of such a study would still be meaningless relative to the effects of ETS on such function in the absence of acceptable verification of exposure and/or dose.

The most widely cited paper purporting to demonstrate an adverse effect of ETS on pulmonary function in adults is the White and Froeb study published in 1980. White and Froeb evaluated adult volunteers enrolled in a fitness program, using a self-administered questionnaire and pulmonary function testing. They noted differences in mid- (FEF_{25-75}) and terminal (FEF_{75-85}) respiratory flow rates between nonsmokers classified as not exposed to ETS and those classified as exposed or passive smokers. White and Froeb have suggested that their study demonstrates that ETS reduces small airways function in the exposed nonsmoker.

Unfortunately, the White and Froeb study suffers from very serious flaws in both design and execution. The study also involves a number of highly questionable assumptions, which undermine the study's validity and White and Froeb's interpretation of their data. Most critically, the White and Froeb study lacks acceptable verification of exposure and relies upon an unvalidated classification of subjects. They used a nonstandardized, nonvalidated, self-derived questionnaire, which their subjects self-administered in an unsupervised fashion. Categorization of ETS exposure based upon inappropriate and poorly designed questionnaires can result in a high degree of misclassification of subjects (Stowijk, et al., 1985; Shy, et al., 1978; Ozkaynak, et al., 1984; Friedman, et al., 1983), which can render totally meaningless any results that are obtained. The use of volunteer subjects also raises a question of selection bias. White and Froeb also failed to assess adequately other environmental exposures and to consider many of the other possible co- and confounding variables described above.

In addition, the equipment used by White and Froeb to test pulmonary function, the Donti Pulmonary Performance Analyzer ("DPPA"), is known to be nonlinear in its response to volume changes and tends to maximize small differences within individual flow volume curves (Lebowitz, 1982). Use of the DPPA therefore is inappropriate for studies such as that undertaken by White and Froeb. In this regard, it is notable that the differences in pulmonary function reported by White and Froeb were in a range no greater than 5-15 percent. Furthermore, the same individual conducted the testing and was responsible for interpretation of the test results, and the testing was done in an unblinded manner, introducing serious questions of investigator bias.

With regard to analysis of the pulmonary function test data, the results reported by White and Froeb were neither age- nor height-adjusted for individuals; instead, statistical comparisons were made on raw flow and volume values, apparently on the assumption that mean age and height were similar for the different groups. Such an assumption is not necessarily valid, since the distribution for ages and heights could very well have been different in different groups in spite of similar averages. In sum, White and Froeb's analysis of the pulmonary function test data generated in their study was, at best, unconventional.

The data reported by White and Froeb showed no significant differences between groups in FVC or FEV_1. In fact, the only differences in pulmonary function parameters reported by White and Froeb were with respect to FEF_{25-75} and FEF_{75-85}. As noted, those differences were quite small --

in the range of 5-15 percent. Furthermore, the actual values remained within the normal range, even in the group with lower values. As indicated previously, whether even abnormally reduced FEF_{25-75} and/or FEF_{75-85} are indicative of small airways disease or dysfunction is open to serious question. To suggest that reduced, but still normal, values for these parameters reflect clinically or physiologically significant dysfunction of the small airways is certainly unjustified.

A study sometimes cited as supporting White and Froeb's conclusions is that of Kauffmann, et al., published in 1983. While Kauffmann and colleagues used a standardized questionnaire, their exposure assessment is, nevertheless, unverified and unvalidated. Although they reported differences in some pulmonary parameters between some groups in some subsets of their study population, which they concluded were consistent with an adverse effect of environmental tobacco smoke on respiratory function, their data are internally inconsistent and cannot be easily interpreted.

For example, the finding by Kauffmann of qualitative and quantitative differences in respiratory function parameters in "passive smoking" men versus "passive smoking" women over the age of 40 is somewhat implausible and is more suggestive of statistical artifact than biological fact. "Light passive" smoking men had FEF_{25-75} values that were superior to those of nonsmoking men, whereas "light" and "intermediate" active smoking men had values for this parameter that were comparable to those of nonsmoking men. Reductions in FEF_{25-75} were reported in "passive" smoking women in the 30-35 year and 40-45 year age groups but not in those over 45 years of age, even though the latter were presumably exposed to ETS for a longer period of time. Additionally, other environmental exposures and other possible co- and confounding variables were not adequately addressed -- including factors such as gas cooking, which could have affected pulmonary function (Lebowitz, 1984a; Comstock, 1981). Finally, even in those groups in which reductions in pulmonary function were noted, the results were still within the normal range and, therefore, cannot validly be said to reflect physiological dysfunction or clinical disease.

Brunekreef and colleagues (Brunekreef, et al., 1985; Remijn, et al., 1985; Fisher, et al., 1985) used a standardized questionnaire, measurement of ambient nitrogen dioxide, and repeated pulmonary function testing as part of a combined cross-sectional and longitudinal study of possible relationships between ETS exposure, gas cooking and pulmonary function in nonsmoking women in rural Holland. Although they used a standardized questionnaire to assess ETS exposure, and their methodology generally was more rigorous than that of many other studies, their estimates of ETS exposure still cannot be said to have been acceptably validated or verified.

The nitrogen dioxide monitoring, for example, was more relevant to gas cooking than ETS exposure.

While Brunekreef reported statistically significant differences between "ETS-exposed" and "nonexposed" subjects in some pulmonary function parameters, the differences were small, and values in both groups were still within the normal range. Furthermore, longitudinal analysis failed to reveal differences between the groups with respect to age-dependent declines in FEV_1 and FVC over time. More significant differences were noted when pulmonary function was related to cooking gas exposure as assessed by nitrogen dioxide measurements. In fact, the authors themselves acknowledged that the differences noted between "ETS-exposed" and "nonexposed" subjects do not necessarily indicate an effect of ETS exposure, and they could not exclude an effect of other variables including in utero and genetic factors.

On the other side of the issue are studies by Comstock, et al. (1981), Kentner, et al. (1984), and Schilling, et al. (1977), which failed to demonstrate an effect of ETS exposure on adult pulmonary function or respiratory symptoms. These studies are also not without significant flaws, not the least of which is a lack of adequate exposure verification. In addition, Comstock and colleagues failed adequately to consider a number of potentially important variables, including occupational and other environmental exposures as well as lifestyle factors. In contrast to their failure to demonstrate an effect of ETS, Comstock did demonstrate an effect of gas cooking. Kentner's data raise questions of sensitivity of the testing techniques used.

Perhaps the most rigorous study is that recently reported by Lebowitz, et al., which addressed the effects of ETS and other indoor and outdoor air pollutants on pulmonary function in 229 subjects from 117 families in Tucson, Arizona (Lebowitz, 1984a; Lebowitz, 1984b; Lebowitz, et al., 1985). While Lebowitz's verification of exposure still leaves something to be desired in that biologic markers of exposure were not measured, his questionnaires were highly standardized, detailed and rigorously applied. Similarly, while all potential co- and confounding variables were not addressed, a larger number of such variables were controlled for in the Lebowitz study than in any other study in the published literature. Although Lebowitz's results cannot be said to be definitive, it is notable that they failed to demonstrate an adverse effect of ETS on pulmonary function or respiratory symptoms in either adults or children, among both asthmatic and nonasthmatic subjects.

With regard to asthma, although anecdotal reports are many, published scientific studies are few and, for the most part, have been poorly done. Dahms, et al. (1981) studied ten individuals with asthma and ten control subjects exposed for one hour to ETS in an exposure chamber. They noted a decline in FVC, FEV_1 and FEF_{25-75} in the asthmatics but not in the controls. Since the subjects were volunteers and five of the ten asthmatics had voiced specific complaints about cigarette smoke prior to the study, the role in this study of psychogenic factors, which are well known to be of importance in asthma (Godfrey & Silverman, 1973; McFadden, et al., 1969; Smith, et al., 1970), is uncertain, a fact that was acknowledged by the authors and may invalidate their conclusions.

Knight and Breslin (1985) studied six asthmatic subjects, performing pulmonary function and histamine challenge tests before and after exposure to mechanically generated smoke. They reported that "passive" smoke exposure produced a decline in pulmonary function and increased histamine reactivity. But Knight and Breslin, like Dahms, failed to control for psychogenic factors, and four of their six subjects gave a history of "ETS-induced" asthma attacks prior to the study. Furthermore, the sequence of testing relative to control and smoke exposure could have influenced the results. Inadequate information regarding their statistical analysis also contributes to largely uninterpretable results.

Other investigators (Shepherd, et al., 1979; Pimm, et al., 1978) have failed to demonstrate an adverse effect of exposure to ETS on asthma. Shepherd's results are most notable in view of his group's attention to random order of control and experimental exposures and the study's extensive pulmonary function testing. Despite the fact that the study involved sufficient smoke exposure to cause mild eye irritation in nine of 14 subjects, and despite the existence of a negative attitude toward cigarette smoking revealed prior to the study by several subjects, no adverse effect of ETS on asthma symptomology was demonstrated.

So far as preexisting chronic obstructive pulmonary disease is concerned, unconfirmed anecdotal reports, both pro and con, abound, especially in the context of public testimony. But a careful review of the scientific literature fails to reveal a single reasonable scientific study that directly addresses the issue of ETS and chronic obstructive pulmonary disease (Higgins, 1985).

In sum, careful review and analysis of the pertinent scientific literature lead to the conclusion that the available data do not support the contention that exposure to environmental tobacco smoke causes or exacerbates pulmonary dysfunction or respiratory disease in adults, at least not on a physiologic or toxicologic basis. That conclusion is consistent with the consensus of scientific opinion as expressed by a 1983 National Institutes of Health Workshop, which concluded: "A review of the data from studies which address the effect of passive smoking on the respiratory system suggests that the effect varies from negligible to quite small" (Public Health Service, 1983).

Respiratory System -- Children

An issue of concern that has been addressed to some extent in the scientific literature is the issue of the possible adverse effects of environmental tobacco smoke ("ETS") on children. This includes, in particular, the possible effects of ETS on pulmonary function, lung development, susceptibility to respiratory disease, and long-term pulmonary health.

Studies addressing the issue of a possible relationship between exposure to ETS and the respiratory health of children need to fulfill the same criteria as studies focusing on adults. In addition, some additional questions are particularly applicable to the pediatric age group, including the following:

1. What is the contribution of ETS to the overall levels of indoor air pollution to which the child is exposed in the home and non-home environments;
2. What are the relevant exposure levels for children to tobacco smoke components in these environments;
3. What are the relationships between exposure levels and delivered doses to children of potentially toxic substances derived from tobacco smoke in these environments;
4. What are the relationships between delivered doses and health effects in children under these circumstances, both short term and long term, and do these differ from those applicable to adults;

5. What are the relative roles and contributions of *in utero* effects of smoking by the mother and environmental tobacco smoke from mother, father and other household members;

6. What are the roles and contributions of genetic and familial factors in any health disturbances noted;

7. What are the roles of diet, parental neglect, transmission of infection from parents and others, socioeconomic factors, hygiene, and other environmental chemicals (*e.g.*, from gas stoves, wood burning appliances, etc.) in any observed health disturbances;

8. What are the roles of other indoor air pollutants (*e.g.*, allergens, fungi, molds, carbon dioxide, nitrogen dioxide, bacteria, lead and other chemicals) in any observed health disturbances;

9. With regard to any observed functional differences between groups, what are the short-term consequences, the long-term consequences, and the natural history of such differences, and what is their biological, physiological and clinical relevance;

10. Do children have unique susceptibilities to tobacco smoke components and/or other indoor environmental substances;

11. Are there different susceptibilities characteristic of different age groups of children; and
12. In older children, has the role of primary and active smoking by the child been adequately taken into account.

To date, most of these questions remain unanswered by the scientific literature.

The majority of the relevant scientific literature in this area has been in the form of epidemiologic studies. Most of these studies have addressed relationships between parental smoking and respiratory health effects and/or pulmonary function in children. All of the published studies to date share a common deficiency -- the absence of adequate verification of exposure or dose. This is true of those studies purporting to show an adverse effect as well as those studies failing to demonstrate such an effect. In the absence of adequate verification of exposure or dose, it is difficult to reach conclusions regarding cause and effect relationships in this or any other area where such relationships are addressed.

One of the largest and most widely publicized epidemiologic studies focusing on the possible effects of parental smoking on pulmonary function in children is a study initiated in 1974 in East Boston, Massachusetts. The East Boston study has, thus far, been published in several phases. The initial phase was published in 1979 (Tager, et al.). The study methodology included the supervised administration of standardized questionnaires dealing with respiratory symptoms and illness, and parental smoking, as well as the measurement of pulmonary function. The initial phase also included a prospective assessment of respiratory illness.

The data from the initial study failed to reveal a statistically significant relationship between pulmonary function in children and the smoking habits of their parents. Furthermore, a 1983 National Institutes of Health Workshop which reviewed the 1979 East Boston and other studies published as of that date pointed out that the type of questionnaire utilized in the East Boston study might not have been entirely suitable since it was designed for other purposes (Public Health Service, 1983). The NIH Workshop also pointed out that "lack of proper attention to the estimation or measurement of exposure is a major weakness of all the studies carried out so far." The 1983 University of Geneva Workshop similarly concluded, after reviewing the 1979 East Boston and other relevant studies, that "no adequate description of ETS dose levels in a population of children has been reported" (Rylander, 1984).

The second phase of the East Boston study was published in 1983 and included a follow-up from the same cohort of children, together with a larger number of subjects who had been followed for seven years using the same techniques (Tager, et al., 1983). This report noted statistically significant lower values for FEV_1 in children with smoking mothers than in children with nonsmoking parents. Paternal smoking was reported to have no effect and no interaction was reported between maternal and paternal smoking. No statistically significant differences were observed with regard to FEF_{25-75}. The authors concluded on the basis of these results that maternal smoking is an important influence on the development of pulmonary function in children, and it was implied that this represented an effect of environmental tobacco smoke.

It should be pointed out that the magnitude of the observed FEV_1 effect in the second phase of the East Boston study was quite small, with an aggregate difference over a five year period of seven percent. Whether such a small functional difference has any clinical significance or relevance remains to be determined. It also is notable that in the group with lower values for FEV_1, the values were still within the normal range.

The East Boston study suffers from a number of significant deficiencies that make interpretation of the data quite difficult. As noted previously, the fact of and amount of exposure to environmental tobacco smoke was based on subjective questionnaire data and lacks adequate verification. There was no measurement of biological markers of ETS exposure. Furthermore, the lack of effect of paternal smoking and the lack of interaction between maternal and paternal smoking on FEV_1 growth suggest the absence of a dose-response relationship. Since an all or none situation relative to this developmental parameter would appear to be scientifically implausible, this casts doubt on the validity of the reported observations. Additionally, a number of potentially significant co- and confounding variables do not appear to have been adequately addressed. These include: other environmental exposures; physical fitness and lifestyle; familial health attitudes and habits; and nutritional factors. It is certainly conceivable that families in which there are no smokers may have fitness and nutrition attitudes and habits that could significantly influence pulmonary function and development.

The third phase of the East Boston study was published in 1984 (Lewitter, et al., 1984). In this phase of the study, the FEV_1 and FEF_{25-75} values that were obtained from parents and children from approximately 400 nuclear families observed over a five-year period were subjected to path analysis in an attempt to identify hereditary and environmental sources of family aggregation. The environmental factors that were considered included respiratory history, nature of home heating, household density and personal smoking habits of parents and children. The analysis revealed that variability in pulmonary function data was influenced to a major degree by genetic heritability and to a lesser degree by common familial environmental effects.

It was further noted that the maternal environment had a consistently greater influence on children's pulmonary function than the paternal environment. On this basis the authors concluded that maternal smoking must influence the pulmonary function of children, a conclusion that implied an adverse effect of environmental tobacco smoke.

It is important to note that this conclusion was derived by inference from the statistical analysis and would appear to go beyond the data. For example, other potentially important maternal factors such as health attitudes and habits, nutrition, lifestyle, etc., still were not included in the analysis. Without attention to such factors, a conclusion that maternal smoking is an important familial environmental factor would appear to be premature.

Three recent epidemiologic studies utilized questionnaires in a format recommended by the epidemiology standardization project of the ATS/DLD to gather information about the smoking habits of occupants and other environmental factors, and also measured pulmonary function. Vedal, et al. (1984) studied 4,000 school children in Western Pennsylvania and observed that maternal smoking reduced some pulmonary function parameters, but not others, in children. Where reductions were reported, they were in the range of 5-8 percent. Paternal smoking had only a trivial effect, while no dose-response relationship was reported between maternal smoking and children's pulmonary function. The effect of maternal smoking was found to be primarily in daughters and not in sons. Ware, et al. (1984) studied more than 10,000 children in the longitudinal Six Cities study. They noted a 0.6-0.9 percent reduction in FEV_1 values of children of smoking mothers when compared to children of nonsmoking mothers. No consistent reduction was noted relative to paternal smoking. A linear relationship was observed relative to number of cigarettes smoked per day by the mother. Finally, Ekwo, et al. (1983) studied 1,355 children from Iowa City. They observed no significant difference in baseline pulmonary function parameters between children with smoking or nonsmoking parents but noted a small enhancement in the response of some parameters to treatment with isoproterenol, which they interpreted to indicate that ETS exposure in children increased airway reactivity to bronchodilators.

Taken together, these three studies based upon the ATS/DLD questionnaire suggest trivial, if any, effects of ETS on pulmonary function in children. The Vedal study failed to demonstrate a dose-response relationship and the demonstration of effects only in females raises questions of plausibility. The Ware, et al., Six Cities study, while appearing to reveal a dose-related effect, deals with very small values -- i.e., 10-20 ml decrements in FEV_1 in the context of total volumes of 1,500-2,500 ml. While statistically significant, it is questionable if such differences have clinical significance or biological relevance. Furthermore, the pulmonary function parameters even in the children exposed to ETS were still within the normal range. The Ekwo study failed to demonstrate an effect of ETS on baseline functional parameters, and the observation of possible enhanced airway reactivity is of uncertain significance and relevance. More importantly, in all three studies the estimates of ETS exposure are unverified and a number of potentially important co- and confounding variables such as lifestyle, health attitudes and habits and nutrition were not adequately addressed. There is also some question as to the validity of the ATS/DLD questionnaire, which was designed to define symptoms of diseases such as chronic bronchitis, in a study of ETS, as was pointed out by the 1983 NIH Workshop (Public Health Service, 1983).

Another recent study by Tashkin, et al. (1984) purports to demonstrate an adverse effect of ETS exposure on pulmonary function in children. However, careful scrutiny of the data reveals potentially invalidating internal inconsistencies such as sex differences and age group differences that are not readily explainable or plausible. In addition, the Tashkin group failed to find a dose-response relationship and the study lacks adequate verification of exposure.

In contrast to these studies purporting to demonstrate an adverse effect, other epidemiologic studies, including those of Schilling, et al. (1977), Dodge (1982), and Hasselblad, et al. (1981), were unable to document significant changes in pulmonary function relative to parental smoking. It is only fair to say, however, that these studies share many of the design deficiencies of those studies purporting to show an effect.

An interesting observation is that of Lebowitz, et al. (1984), who evaluated data from 271 nuclear families in the Tucson epidemiologic study on airway obstructive diseases. Lebowitz's data indicate familial aggregation of pulmonary function, with particular aggregation relative to body habitus, and suggest that genetically determined factors may be more important than had previously been appreciated. Lebowitz was unable to confirm a significant effect of parental smoking on children's pulmonary function, although his study can also be criticized because of inadequate exposure verification. Lebowitz, et al. have also demonstrated that household aggregation of pulmonary function is primarily due to household aggregation of body size (Lebowitz, et al., 1982b).

A number of epidemiologic studies in the literature have addressed respiratory symptoms and illness. These are generally less reliable than pulmonary function parameters, and symptom reporting in children may reflect parental biases (Lebowitz, et al., 1982b; Cederloff and Colley, 1974; Lebowitz and Burrows, 1976; Schilling, et al., 1977). Furthermore, like the studies addressing pulmonary function, all such studies also suffer from the major potentially invalidating deficiency of lack of adequate verification of exposure or dose.

The report of Bland, et al. (1978) purportedly demonstrating a parental smoking effect on respiratory symptoms and illness in children is significantly flawed by the failure to supervise completion by the child subjects of the study questionnaires, absence of validation of ETS exposure, and failure to consider important other variables. The study of Bonham and Wilson (1981) failed to address major co- and confounding variables, in addition to lacking verified exposure estimates. Fergusson, et al. (1981) reported an effect of maternal smoking on rates of lower respiratory illness only during the first year of life, with that effect disappearing during the second and third years of life, an observation more consistent with an in utero or lactational effect than an ETS effect. Furthermore, in addition to lacking adequate exposure verification, a number of potentially confounding variables were not taken into account in the Fergusson study.

A recent report by Pedreira, et al. (1985) failed to use even standardized respiratory questionnaires, ignored major potentially confounding variables, failed to obtain qualitative smoking information on 40 percent of the parents, and contains a number of internal inconsistencies, as well as lacking a dose-response relationship in some groups.

Gortmaker, et al. (1982), relied on data obtained by telephone survey, with diagnoses based on parental response without objective examination of the children, techniques that for all practical purposes invalidate the data. Studies by Cameron, et al. (1973) contain major internal inconsistencies that have been commented upon in a report of the Surgeon General (1979). As also noted in that report, studies by Colley, et al. (1974) provide data that are at least as consistent with transmission of infectious agents from parents as an ETS effect. While Harlap and Davies (1974) found a relationship between admissions for respiratory illness in the first year of life and maternal smoking habits during pregnancy, they found a similar relationship between maternal smoking and infant admissions for poisoning and injuries, suggesting a relationship with parental neglect or socioeconomic class rather than ETS.

In contrast to these other studies, Lebowitz, et al. (1982b) failed to demonstrate a relationship between parental smoking and present or past symptoms, including persistent wheeze or early childhood lower respiratory illness.

In conclusion, an association between environmental tobacco smoke and pulmonary dysfunction or disease in children remains to be convincingly demonstrated. The available data are inconsistent and contradictory and where purportedly positive effects are observed, they are invariably small in magnitude, still within the normal range, and are of uncertain clinical or biological significance or relevance. All of the published studies suffer from the major potentially invalidating deficiency of lack of verification of ETS exposure or dosage, and many of the studies fail adequately to address familial or genetic aggregation of pulmonary function parameters. Epidemiologic studies focusing on symptoms and illness are similarly flawed and difficult to interpret, as well as often internally inconsistent and contradictory. In none of the pertinent studies was adequate account taken of possible in utero or lactational effects as opposed to ETS effects, and none of the published studies adequately addressed nutritional status, lifestyle and familial health habits and attitudes.

Cardiovascular System

Carbon monoxide and nicotine are two components of tobacco smoke that have pharmacologic properties which may affect the cardiovascular system. Nicotine can stimulate the release of catecholamines from the sympathetic nervous system and the adrenal medulla. It also can directly affect the vasomotor center in the brain, and in so doing may affect heart rate and blood pressure. Carbon monoxide binds to hemoglobin 200 times more avidly than does oxygen and may interfere with hemoglobin function by reducing the oxygen binding capacity of hemoglobin. In addition, it can cause a leftward shift in the oxyhemoglobin dissociation curve.

It has been suggested that the combination of nicotine and carbon monoxide may be particularly inimicable to myocardial function, since nicotine may result in increased myocardial oxygen demand while carbon monoxide may decrease the availability of oxygen to the myocardium. It has been further suggested that individuals with preexisting coronary artery disease and impaired myocardial blood flow may be particularly vulnerable to the adverse effects of nicotine and carbon monoxide. Since carbon monoxide and nicotine are significant constituents of tobacco smoke, and by extrapolation from data relative to active smoking, it has been postulated that environmental tobacco smoke may have adverse effects on cardiac function, particularly in patients with preexisting coronary artery disease.

While such a hypothesis is at first glance logical and plausible, it is not necessarily a foregone conclusion. The effects of nicotine and carbon monoxide on cardiac function are dependent upon dose, and the occurrence of adverse cardiovascular effects would require exposure to a sufficient concentration of these chemicals to result in a delivered dose that would exceed the threshold for toxicity. In view of the quantitative and likely qualitative differences between environmental tobacco smoke, mainstream smoke, and sidestream smoke, and the multiplicity and complexity of factors (including a number that are as yet undetermined) affecting the concentration of these chemicals in the ambient environment, it is necessary to establish that environmental tobacco smoke results in such potentially toxic concentrations of nicotine and/or carbon monoxide and/or to demonstrate that environmental tobacco smoke indeed adversely affects cardiac function and the cardiovascular system. Theoretical plausibility is unquestionably a requirement for determining a cause-and-effect relationship between a chemical substance and adverse health effects. But it is not by itself an adequate substitute for studies, focusing specifically on environmental tobacco smoke, designed and executed in accordance with accepted scientific criteria.

Although statements frequently have been made in both scientific and lay literature that environmental tobacco smoke adversely affects cardiovascular function and the cardiovascular system of exposed nonsmokers, after careful and thorough review of the pertinent literature, it is apparent that the available scientific data do not reliably support such a conclusion. This finding is consistent with the mainstream of considered scientific opinion, as reflected in the conclusions of all major groups that similarly have reviewed the pertinent data, including the major international scientific workshops that have been convened to assess the possible health effects of exposure to environmental tobacco smoke (Rylander, 1984; Lehnert and Wynder, 1984).

The study that is cited most frequently to support the contention that exposure to ETS is hazardous to cardiovascular health was published by Aronow in 1978. Aronow examined the effects of "passive" smoking on exercise-induced angina in men with stable exertional angina and angiographic evidence of severe coronary artery disease. He concluded that exposure to environmental tobacco smoke reduced the duration of exercise necessary to produce angina, and suggested that this was the result of a combination of the adverse effects of carbon monoxide and nicotine.

A careful examination of Aronow's data reveals a number of problems that make interpretation difficult, at best, and raises considerable doubt about the validity of his results and conclusions. In particular, the Aronow study can be criticized for the use of the unconventional endpoint of subjective pain as a criterion for angina, rather than the more acceptable and standard criterion of ST-segment depression. That criticism is particularly compelling in the case of the Aronow study, which was not double blinded. Furthermore, psychogenic and emotional factors, generally considered to be especially important in patients with coronary artery diseases, were neither appropriately nor adequately addressed in the Aronow study. Despite the importance Aronow attached to carbon monoxide, ambient levels of this chemical were not measured. While blood levels were obtained, the theoretical increases in ambient carbon monoxide under the conditions of his study would have been insufficient to produce the reported increases in carboxyhemoglobin that Aronow noted. In addition, the relationship between blood levels of carboxyhemoglobin and impaired work performance were inconsistent with other data in the literature, including data previously reported by Aronow (Aronow and Isbell, 1973; Aronow, et al., 1972; Aronow, et al., 1974). In this regard, it is pertinent to note that the general reliability and validity of data reported by Aronow, including data relative to carbon monoxide and carboxyhemoglobin, have been seriously questioned by two federal agencies (Bodiansky, 1983; Schievelbein and Richter, 1984).

With regard to Aronow's suggestions with respect to nicotine, it should be noted that levels of nicotine or nicotine metabolites were not determined -- and it is not likely, on theoretical grounds, that Aronow's subjects received sufficient doses of nicotine to result in adverse cardiovascular effects. Other significant criticisms of Aronow's study include inadequate controls and flawed and inappropriate statistical methodology, as well as failure to address a potential role of carbon dioxide build-up under the conditions of his study. In sum, the results of the Aronow study are frankly not credible and the conclusions of its author are not justified.

Hurschman, et al. (1978) reported that a five-minute exposure to environmental tobacco smoke increased heart rate and systolic blood pressure at rest and potentiated an increase in heart rate and blood pressure during treadmill exercise in both smoking and nonsmoking women. They further reported that these effects were more pronounced in nonsmokers than in smokers and that, during exercise, blood carboxyhemoglobin increased 1.5 percent in nonsmokers while decreasing 1 percent in smokers. They concluded from these observations that nonsmokers were less tolerant than smokers of environmental tobacco smoke and that passive smoke exposure, by virtue of its hemodynamic effects, was potentially hazardous to the elderly, the untrained, and those with cardiac disease.

There are a number of problems with Hurschman's study. Although Hurschman and colleagues may have observed a physiological effect, such observation does not necessarily translate into an adverse health effect. Furthermore, their data are inadequate to establish a cause-and-effect relationship between smoke exposure _per se_ and any physiological event. In addition, it is uncertain from their published data if they indeed observed any significant hemodynamic effect at all. The mean differences in the measured parameters between their groups were quite small and estimates of variability, such as standard deviations, were not provided. Although the authors state that the data were analyzed by ANOVA and Duncan's New Multiple Range Test, no details of the analyses are given, and it is uncertain whether the data were, in fact, processed appropriately. In this regard, it is notable that Hurschman, et al. claimed statistical significance at a p value of less than 10, which is a meaningless statistical term.

Moreover, the reported increase in carboxyhemoglobin of 1.5 percent in nonsmokers seems implausibly high for a 15-minute exposure to an environment in which the ambient carbon monoxide was stated to be 20 parts per million. This suggests the possibility that either their ambient carbon monoxide or blood carboxyhemoglobin measurements were in error. Theoretically, a 15 minute exposure during exercise to an ambient carbon monoxide of 20 parts per million should elevate the carboxyhemoglobin about 0.15 percent, not 1.5 percent (Turino, 1981). An increase in carboxyhemoglobin of 1.5 percent would require exposure to an ambient carbon monoxide level of 200 parts per million. Additionally, the authors do not adequately exclude the possibility that the hemodynamic effects they observed were a physiologic response to the emotional and psychological conditions of the experiment rather than a pharmacologic response to components of environmental tobacco smoke.

In contrast to the reports by Aronow and Hurschman, et al., other studies in which subjects were exposed to environmental tobacco smoke, with carbon monoxide levels ranging from 60 parts per million for 20 minutes to 24 parts per million for two hours, failed to find any effect on heart rate or blood pressure (Schievelbein and Richter, 1984).

In a recently reported epidemiologic study, Garland, et al. (1985) attempted to examine the relationship between death from ischemic heart disease in nonsmoking women and the smoking habits of their spouses. They evaluated a group of women classified according to their husband's self-reported smoking status as "never smoked," "former smoker," and "current smoker." While they could find no significant difference in overall death rates among the three groups, when they combined the wives of "current smokers" with the wives of "former smokers" into a single group, they found a significantly greater mortality from ischemic heart disease in this group compared to the wives of "never smokers." They suggested that this increased ischemic heart disease mortality was due to "passive smoking."

The Garland study suffers from a number of major, potentially invalidating flaws that render both its findings and conclusions open to serious question. There are, for example, very serious problems with the design and the statistical/epidemiological methodology used in the Garland study. These include the fact that the subjects were volunteers who agreed to participate, thus raising the question of potential self-selection bias, and 18 percent of those solicited refused to participate, which might have been sufficient to alter the outcome and conclusions. The fact that two heterogeneous groups, wives of "former smokers" and wives of "current smokers" were pooled is anomalous and inappropriate. Furthermore, the authors chose to analyze mortality rather than the more conventional analysis of survival. In fact, if one analyzes survival rather than mortality and treats the three groups separately, the results are contrary to those reported by the authors. This suggests that wives of "current smokers" have longer survival rates than even wives of "former smokers" or "never smokers." Thus, it is quite probable that the results reported by Garland and his colleagues were simply an artifact of the analysis.

A further deficiency in the Garland study was the investigators' failure to verify exposure. In addition, no data were provided about the duration of the husband's smoking or about how much smoking actually occurred in the home. Major potentially confounding variables also were not adequately addressed, including such critical variables relative to cardiovascular disease as lifestyle, diet and exercise, as well as factors such as husband's occupation, genetic factors, familial factors, and intrinsic diseases related to cardiovascular disease and mortality such as diabetes mellitus. It should also be noted that the authors were unable to demonstrate a statistically significant dose-response relationship between the number of cigarettes smoked by husbands and their wives' death rate.

The conclusions of Garland and some other investigators presuppose that chronic exposure to environmental tobacco smoke is an important factor in the pathogenesis of coronary artery disease. But animal studies have failed to demonstrate an atherogenic effect of carbon monoxide or nicotine (Schievelbein and Richter, 1984). Furthermore, as has been pointed out by Schievelbein and Richter (1984), the suggestion of a causal relationship between exposure to environmental tobacco smoke and coronary heart disease is doubtful in light of the fact that, in the Framingham and other studies of female active smokers, the association between cigarette smoking and coronary heart disease was found to be much weaker than in male smokers and, in fact, could not be convincingly demonstrated (Dawber, 1980; Kannel, 1981). This makes it somewhat implausible to assume that such an effect occurs in female "passive smokers."

Since carbon monoxide and nicotine have been suggested as possible pathogenetic factors for the putative adverse cardiovascular effects of environmental tobacco smoke, it is pertinent to examine the data relative to the contribution of environmental tobacco smoke to levels of these indoor air pollutants and their uptake by exposed nonsmokers. In studies of indoor carbon monoxide levels relative to smoking, Bridge and Corn (1972) concluded that measured and predicted carbon monoxide concentrations from smoking in occupied spaces are generally below federal ambient air quality standards and do not present a hazard to nonsmokers. Olshansky (1982) estimated increases in carboxyhemoglobin concentration from exposure to smoking in real life situations to be in the range of 0.5 to 0.8 percent for a three hour exposure. Cole (1975) noted mean blood carboxyhemoglobin concentrations of 1.12 percent in nonsmokers working in an indoor office environment where smoking was permitted. Szadowski, et al. (1976) reported average blood carboxyhemoglobin concentrations of nonsmokers exposed to smoke in offices to be consistently below 1 percent and no significant differences in blood levels between nonsmokers from offices with little or no tobacco smoke and those considered to have high tobacco smoke levels.

In an experimental study in which nonsmokers were exposed to smoking in an unventilated room, Russell and Feyerabend (1975) noted only modest increases in plasma nicotine concentration -- i.e., from 0.73 nanograms per ml to 0.90 nanograms per ml, associated with an eight-fold increase in urinary nicotine. They concluded that nicotine is excreted almost as rapidly as it is absorbed and that this rapid excretion accounts for an absence of obvious physiological effects of smoke exposure on nonsmokers. Other studies in the literature have tended to be consistent with Russell and Feyeraband's findings (Schievelbein and Richter, 1984).

In conclusion, the available data fail to support either the actuality or the plausibility of an adverse effect of environmental tobacco smoke on cardiovascular function in the nonsmoker. The laboratory studies of Aronow and Hurschman that claim such an association are sufficiently flawed in design and methodology to be of no validity relative to this issue. The epidemiologic study of Garland, et al. is similarly deficient in design and methodology and thus is not valid nor to the point. Under real life conditions, the contribution of environmental tobacco smoke to carbon monoxide and nicotine levels in the ambient air is minimal and the uptake and retention of those constituents appear to be insufficient to produce a pathophysiologic effect.

References

American Thoracic Society: Report of Snowbird Workshop on Standardization of Spirometry. Am. Rev. Respir. Dis. 119:831-38, 1979.

Aronow, W.S.: Effect of Passive Smoking on Angina Pectoris. N. Engl. J. Med. 299:21-4, 1978.

Aronow, W.S. and Isbell, M.W.: Carbon Monoxide Effect on Exercise-Induced Angina Pectoris. Ann. Intern. Med. 79:392-5, 1973.

Aronow, W.S., et al.: Effect of Cigarette Smoking and Breathing Carbon Monoxide on Cardiovascular Hemodynamics in Anginal Patients. Circulation 50:340-7, 1974.

Aronow, W.S., et al.: Effect of Freeway Travel on Angina Pectoris. Ann. Intern. Med. 77:699-76, 1972.

Bates, D.V.: The Fate of the Chronic Bronchitic: A Report of the Ten-Year Follow Up in the Canadian Department of Veterans Affairs Coordinated Study of Chronic Bronchitis. Am. Rev. Respir. Dis. 108:1043-65, 1973.

Berend, N. and Thurlbeck, W.M.: Correlations of Maximum Expiratory Flow With Small Airway Dimensions and Pathology. J. Appl. Physiol. 52:346-51, 1982.

Beus, M.L., et al.: Hazards of Using FEF25-75 as Indicator of Obstructive Lung Disease. Am. Rev. Respir. Dis. 123 (part 2):102 (abst.), 1981.

Bland, M., et al.: Effect of Children's and Parents' Smoking on Respiratory Symptoms. Arch. Dis. Child. 53:100-5, 1978.

Bodiansky, S.: Data Falsification: Food and Drug Data Fudged. Nature (London) 302:560, 1983.

Bonham, G.S. and Wilson, R.W.: Children's Health in Families With Cigarette Smokers. Am. J. Public Health 71:290-3, 1981.

Brain, J.D. and Barry, B.E.: Biological Potential and Exposure-Dose Relationships for Constituents of Cigarette Smoke, in Gammage, R.B., et al. (eds.). Indoor Air and Human Health, Lewis Publishers, pp. 215-25, 1985.

Bridge, D.P. and Corn, M.: Contribution of the Assessment of Exposure of Nonsmokers to Air Pollution from Cigarette and Cigar Smoke in Occupied Spaces. Environ. Res. 5:192-209, 1972.

Brunekreef, B., et al.: Indoor Air Pollution and Its Effect on Pulmonary Function of Adult Nonsmoking Women: III. Passive Smoking and Pulmonary Function. Internat. J. Epidemiol. 14: 227-30, 1985.

Buist, A.S. and Ducic, S.: Smoking. Evaluation of Studies Which Have Demonstrated Pulmonary Function Changes, in Macklem, P.T.. Permutt, S. (eds.). The Lung in the Transition Between Health and Disease, Vol. 12: Lung Biology in Health and Disease, New York: Marcel Dekker, 1979.

Burrows, B., et al.: Respiratory Disorders. A Pathophysiologic Approach. Yearbook Medical Publishers, 1983.

Cameron, P., et al.: Effect of Home Environment Tobacco Smoke on Family Health. J. Appl. Physiol. 67:142-57, 1973.

Cederloff, R. and Colley, J.: Epidemiological Investigations on Environmental Tobacco Smoke. Scand. J. Respir. Dis. Suppl. 91:47-49, 1974.

Cole, P.V.: Comparative Effects of Atmospheric Pollution and Cigarette Smoking on Carboxyhemoglobin Levels in Man. Nature 255:699-701, 1975.

Colley, J.R.T., et al.: Influence of Passive Smoking and Parental Phlegm on Pneumonia and Bronchitis in Early Childhood. Lancet 2:1031-34, 1974.

Comstock, G.W., et al.: Respiratory Effects of Household Exposures to Tobacco Smoke and Gas Cooking. Am. Rev. Respir. Dis. 124:143-48, 1981.

Cosio, M.G., et al.: The Relations Between Structural Changes in Small Airways and Pulmonary Function Tests. N. Engl. J. Med. 298:1277-81, 1977.

Dahms, T.E., et al.: Passive Smoking: Effects on Bronchial Asthma. Chest 80:530-34, 1981.

Dawber, T.R.: The Framingham Study. The Epidemiology of Atherosclerotic Disease. Harvard Univ. Press, Cambridge, Mass., 1980.

Dodge, R.: The Effects of Indoor Pollution on Arizona Children. Arch. Envir. Health 37:151-55, 1982.

Ekwo, E.E., et al.: Relationship of Parental Smoking and Gas Cooking to Respiratory Disease in Children. Chest 84:662-68, 1983.

Fergusson, D.M., et al.: Parental Smoking and Lower Respiratory Illness in the First Three Years of Life. J. Epidemiol. Community Health 35:180-84, 1981.

First, M.W.: Constituents of Sidestream and Mainstream Tobacco Smoke and Markers to Quantify Exposure to Them, in Gammage, R.B., et al. (eds.). Indoor Air and Human Health, Lewis Publishers, pp. 195-203, 1985.

Fisher, P., et al.: Indoor Air Pollution and Its Effect on Pulmonary Function of Adult Nonsmoking Women: II. Associations Between Nitrogen Dioxide and Pulmonary Function. Internat. J. Epidemiol. 14:221-26, 1985.

Fletcher, C.M., et al.: The Natural History of Chronic Bronchitis and Emphysema. New York: Oxford University Press, 1976.

Friedman, G.D., et al.: Prevalence and Correlates of Passive Smoking. Am. J. Public Health 73:401-05, 1983.

Garland, C., et al.: Effects of Passive Smoking on Ischemic Heart Disease: Mortality of Nonsmokers -- A Prospective Study. Am. J. Epidemiol. 121:645-50, 1985.

Godfrey, S. and Silverman, M.: Demonstration of Placebo Response in Asthma by Means of Exercise Testing. J. Psychosom. Res. 17:293-97, 1973.

Gortmaker, S.L., et al.: Parental Smoking and the Risk of Childhood Asthma. Am. J. Public Health 72:574-79, 1982.

Harlap, S. and Davies, A.M.: Infant Admissions to Hospital and Maternal Smoking. Lancet 1:529-32, 1974.

Hasselblad, V., et al.: Indoor Environmental Determinants of Lung Function in Children. Am. Rev. Respir. Dis. 123:479-85, 1981.

Higgins, M.: Critical Review of the Relationship Between Passive Exposure to Cigarette Smoke and Cardiopulmonary Disease, in Gammage R.B., et al. (eds.). Indoor Air and Human Health, Chelsea, MI, Lewis Publishers, pp. 241-56, 1985.

Hurschman, L.G., et al.: The Implications of Sidestream Cigarette Smoke for Cardiovascular Health. J. Environ. Health 41:145-49, 1978.

Johnston, R.N., et al.: Chronic Bronchitis: Measurements and Observations Over 10 Years. Thorax 31:25-29, 1976.

Kannel, W.B.: Update on the Role of Cigarette Smoking in Coronary Artery Disease. Am. Heart J. 101:319-28, 1981.

Kauffmann, F., et al.: Adult Passive Smoking in the Home Environment: A Risk Factor for Chronic Airflow Limitation. Am. J. Epidemiol. 117:269-80, 1983.

Kentner, M., et al.: The Influence of Passive Smoking on Pulmonary Function--A Study of 1351 Office Workers. Prev. Med. 13:656-69, 1984.

Knight, A. and Breslin, A.B.: Passive Cigarette Smoking and Patients With Asthma. Med. J. Austral. 142:194-95, 1985.

Lebowitz, M.D.: Letter to Chairman, Subcommittee on Tobacco and Peanuts, House of Representatives. Congressional Record, pp. E5253-54, December 16, 1982.

Lebowitz, M.D.: The Effects of Environmental Tobacco Smoke Exposure and Gas Stoves on Daily Peak Flow Rates in Asthmatic and Nonasthmatic Families. Eur. J. Respir. Dis. 65 (suppl. 133):90-97, 1984a.

Lebowitz, M.D.: Influence of Passive Smoking on Pulmonary Function: A Survey. Prev. Med. 13:645-55, 1984b.

Lebowitz, M.D.: Utilization of Data from Human Population Studies for Setting Air Quality Standards: Evaluation of Important Issues. Environ. Health Perspectives, 52:193-205, 1983.

Lebowitz, M.D., et al.: Significance of Intraindividual Changes in Maximum Expiratory Flow Volume and Peak Expiratory Flow Measurements. Chest 81:566-70, 1982a.

Lebowitz, M.D., et al.: Respiratory Symptoms and Peak Flow Associated With Indoor and Outdoor Air Pollutants in the Southwest. JAPCA 35:1154-58, 1985.

Lebowitz, M.D., et al.: Family Aggregation of Pulmonary Function Measurements. Am. Rev. Respir. Dis. 129:8-11, 1984.

Lebowitz, M.D., et al.: The Effect of Passive Smoking on Pulmonary Function in Children. Environ. Internat. 8:371-73, 1982b.

Lebowitz, M.D. and Burrows, B.: Respiratory Symptoms Related to Smoking Habits of Family Adults. Chest 69:48-50, 1976.

Lehnert, G. and Wynder, E.: Medical Perspective on Passive Smoking. Prev. Med. 13:557-746, 1984.

Lewitter, F.I., et al.: Genetic and Environmental Determinants of Level of Pulmonary Function. Amer. J. Epidemiol. 120:518-30, 1984.

McFadden, E.R. and Ingram, R.H.: Clinical Application and Interpretation of Airway Physiology, in Nadel, J.A. (ed.). Physiology and Pharmacology of the Airways. New York: Marcel Dekker, pp. 297-323, 1980.

McFadden, E.R., et al.: The Mechanism of Action of Suggestion in the Induction of Acute Asthma Attacks. Psychosom. Med. 31:134-43, 1969.

Meadows, J.A., et al.: Density Dependence of Maximal Expiratory Flow in Chronic Obstructive Pulmonary Disease. Am. Rev. Respir. Dis. 121:47-53, 1980.

Miller, M.R., Grove, D.M. and Pincock, A.C.: Time Domain Spirogram Indices: Their Variability and Reference Values in Nonsmokers. Am. Rev. Respir. Dis. 132:1041-48, 1985.

Miller, M.R., Pincock, A.C. and Grove, D.M.: Patterns of Spirogram Abnormality in Individual Smokers. Am. Rev. Respir. Dis. 132:1034-40, 1985.

Mink, S.N. and Wood, L.D.H.: How Does HeO2 Increase Maximum Expiratory Flow in Human Lungs? J. Clin. Invest. 66:720-29, 1980.

Morris, A.H., et al. (eds.): Clinical Pulmonary Function Testing. A Manual of Uniform Laboratory Procedures. Intermountain Thoracic Society, 1984.

Nagai, A., et al.: The National Institutes of Health Intermittent Positive-Pressure Breathing Trial: Pathology Studies. II. Correlation Between Morphologic Findings, Clinical Findings, and Evidence of Expiratory Air-Flow Obstruction. Am. Rev. Respir. Dis. 132:946-53, 1985.

Nemery, B., et al.: Significance of Small Airway Tests in Middle-Aged Smokers. Am. Rev. Respir. Dis. 124:232-38, 1981.

Olshansky, S.J.: Is Smoker/Nonsmoker Segregation Effective in Reducing Passive Inhalation Among Nonsmokers? Am. J. Public Health 72:737-39, 1982.

Ozkaynak, H., et al.: Bias Due to Misclassification of Personal Exposures in Epidemiological Studies of Indoor and Outdoor Air Pollution. Proc. 3rd Internat. Conf. on Indoor Air Quality and Climate, Stockholm, 1984.

Pedreira, F.A., et al.: Involuntary Smoking and Incidence of Respiratory Illness During the First Year of Life. Pediat. 75:594-97, 1985.

Pimm, P.E., et al.: Physiologic Effects of Acute Passive Exposure to Cigarette Smoke. Arch. Environ. Health 33:201-13, 1978.

Public Health Service. Report of a Workshop on Respiratory Effects of Involuntary Smoke Exposure: Epidemiologic Studies. United States Department of Health and Human Services, Dec. 1983.

Remijn, B., et al.: Indoor Air Pollution and Its Effect on Pulmonary Function of Adult Nonsmoking Women: I. Exposure Estimates for Nitrogen Dioxide and Passive Smoking. Internat. J. Epidemiol. 14:215-20, 1985.

Russell, M.A.H. and Feyerabend, C.: Blood and Urinary Nicotine in Nonsmokers. Lancet 1:179-81, 1975.

Rylander, R. (ed.): ETS-Environmental Tobacco Smoke: Report of a Workshop on Effects and Exposure Levels. Eur. J. Respir. Dis. 65 (suppl. 133):1-152, 1984.

Schievelbein, G. and Richter, F.: The Influence of Passive Smoking on the Cardiovascular System. Prev. Med. 13:626-44, 1984.

Schilling, R.S.F., et al.: Lung Function, Respiratory Disease, and Smoking in Families. Am. J. Epidemiol. 106:274-83, 1977.

Shepherd, R.J., et al.: Passive Exposure of Asthmatic Subjects to Cigarette Smoke. Environ. Res. 20:392-402, 1979.

Shy, C.M., et al.: The Effect of Misclassification of Exposure Status in Epidemiological Studies of Air Pollution Health Effects. Bull. N.Y. Acad. Med. 54:1155-65, 1978.

Smith, M.M., et al.: Increase and Decrease in Pulmonary Resistance With Hypnotic Suggestion in Asthma. Am. Rev. Respir. Dis. 102:236-42, 1970.

Snow, M.: Instrumentation, in Clausen, J.L. (ed.). Pulmonary Function Testing Guidelines and Controversies: Equipment, Methods and Normal Values. Academic Press, pp. 27-47, 1982.

Stowijk, J.A.J., et al.: Experimental Considerations in the Measurement of Exposures to Sidestream Cigarette Smoke, in Gammage, R.B., et al. (eds.). Indoor Air and Human Health, Lewis Publishers, pp. 205-13, 1985.

Suratt, P.M., et al.: Effect of Maximal Versus Submaximal Expiratory Effort on Spirometric Values. Respiration 42:233-36, 1981.

Surgeon General: Report of the Surgeon General on Smoking and Health, U.S. Public Health Service, 1979.

Szadowski, D., et al.: Body Burden of Carbon Monoxide from Passive Smoking in Offices. Inn. Med. 3:310-13, 1976.

Tager, I.B., et al.: Effect of Parental Cigarette Smoking on the Pulmonary Function of Children. Am. J. Epidemiol. 110: 15-26, 1979.

Tager, I.B., et al.: Longitudinal Study of the Effects of Maternal Smoking on Pulmonary Function in Children. N. Engl. J. Med. 309:699-703, 1983.

Tashkin, D.P., et al.: The UCLA Population Studies of Chronic Obstructive Respiratory Disease. VII. Relationship Between Parental Smoking and Children's Lung Function. Am. Rev. Respir. Dis. 129:891-97, 1984.

Tattersall, S.F., et al.: The Use of Tests of Peripheral Lung Function for Predicting Future Disability from Airflow Obstruction in Middle-Aged Smokers. Am. Rev. Respir. Dis. 118:1035-50, 1978.

Turino, G.M.: Effect of Carbon Monoxide on the Cardiorespiratory System. Circulation 63:253A-59A, 1981.

Vedal, S., et al.: Risk Factors for Childhood Respiratory Disease. Am. Rev. Respir. Dis. 130:187-92, 1984.

Vollmer, W.M., et al.: Relationship of Response to a Bronchodilator and Decline in Forced Expiratory Volume in One Second in Population Studies. Am. Rev. Respir. Dis. 132:1186-93, 1985.

Ware, J.H., et al.: Passive Smoking, Gas Cooking and Respiratory Health in Children Living in Six Cities. Am. Rev. Respir. Dis. 129:366-74, 1984.

White, J.R. and Froeb, H.R.: Small-Airways Dysfunction in Nonsmokers Chronically Exposed to Tobacco Smoke. N. Engl. J. Med. 302:720-23, 1980.

Paper No. 86-80.9

Causal Relationship Between Environmental Tobacco Smoke and Lung Cancer in Non-smokers: A Critical Review of the Literature[1]

Nancy J. Balter, Ph.D.
Department of Biology
Georgetown University
Washington, D.C.

S. James Kilpatrick, Ph.D.
Department of Biostatistics
Medical College of Virginia
Richmond, Virginia

Philip Witorsch, M.D.
Department of Medicine
The George Washington University
Washington, D.C.

Sorell L. Schwartz, Ph.D.
Department of Pharmacology
Georgetown University
Washington, D.C.

---

[1] Tentatively accepted for publication, Proc. Air Pollution Control Association

Introduction

Recent publications in both the scientific and lay press have focused on the question of a relationship between exposure to environmental tobacco smoke (ETS) and lung cancer. There are two current questions. Do the existing data establish that an association exists between ETS exposure and lung cancer? If not, what evidence would be necessary to establish causation? The answer to the first question in part depends upon how one approaches the answer to the second, i.e. the basis for establishing causation. There are three apparent approaches to determining whether or not a causative relationship exists between exposure to ETS and the incidence of lung cancer.

1. Extrapolate from active smoking data.

There are significant problems with this approach, not the least of which is the *a priori* assumption of causation based on a relationship of ETS exposure to active smoking. Environmental tobacco smoke is a term used to describe a complex and dynamic mixture of exhaled mainstream and sidestream tobacco smoke. Sidestream smoke (SS) refers to the mixture of chemicals released into the air from the burning tip of the cigarette. The contribution of SS to ETS predominates, accounting for 80-90% of the total release of most chemicals[1]. The mixture of exhaled mainstream smoke and SS which comprises ETS is dynamic with respect to time, undergoing both physical and chemical changes referred to as aging. Among the most significant contributors to the aging process are dilution in room (or ambient) air and removal by ventilation, filtration and non-specific adherence to surfaces.

Attempts have been made to characterize the chemical composition of SS. Hoffmann and his coworkers, among others, have provided data on the relative concentration of a number of chemicals in sidestream versus mainstream tobacco smoke (see 2 for review). As would be expected, considering the lack of filtration of SS compared to MS, SS:MS ratios are often greater than 1.

In addition to the differences in chemical composition between ETS and MS, there are several toxicologically significant differences with respect to exposure. Exposure to ETS involves essentially constant low-dose exposure occurring primarily by inhalation through the nose. By comparison, the active smoker receives intermittent, high-dose exposure primarily through the mouth. Active smokers characteristically inhale more deeply and may pause before exhaling. Such differences would be reflected in substantial discrepancies between active and "passive" smokers in the regional deposition and retention of tobacco smoke constituents[3-4] and, thus, in the delivered dose of chemicals to various sites in the respiratory tract. Consequently, estimation of ETS exposure in terms of "cigarette equivalents" is misleading and unjustified on a scientific basis.

Based on a consideration of these differences in the exposure of the active smoker to MS and exposure of the non-smoker to ETS, it is logical to conclude that the two represent toxicologically distinct phenomena[5]. Therefore, the contribution of exposure to ETS as a risk factor for lung cancer cannot be extrapolated from active smoking data.

This leads to the second possible approach to the question of ETS and lung cancer causastion.

2. Extrapolate based on the presence of individual carcinogens in ETS.

Again, this is an approach which assumes _a priori_ that ETS can cause lung cancer, in this case based upon the presence of substances which are individually classed as carcinogens based on the results of animal bioassays. The demonstration of chemical carcinogens in SS does not, by itself, imply an increased risk of lung cancer. Whether or not the lung tissue of an individual actually receives a dose of carcinogen depends upon a variety of factors including the physical and chemical aging of SS in room air and the characteristics of the exposure of the individual. With respect to the former, despite the often-quoted literature on SS:MS ratios, relatively little direct data exist on the chemical composition of ETS and the influence of aging on its composition. Of the studies which have been purported to examine the chemical composition of ETS, the majority report the concentrations of various chemicals in indoor air in the presence of smoking. However, with few exceptions, the chemicals that have been measured are not unique to ETS and the background (non-smoking condition) levels of these contaminants are inadequately reported.

The study of Brunnemann and Hoffmann[6,7] is typical. In this study dimethylnitrosamine (DMN), an animal carcinogen, was detected in air samples collected from rooms containing smokers. The smoke density was obviously great, as the authors report that the aerosol was visible and the individuals taking the samples experienced eye irritation. However, the experimental design does not allow one to identify ETS as the source of the DMN since no comparable measurements were made in the absence of smoking. Based on a review of Brunnemann and Hoffmann's data as well as their own, Stehlik _et al._[8] have concluded that additional sources of DMN must have been present.

The absolute requirement for establishing background levels of chemicals with competing sources in order to assess the contribution of any single source, such as ETS, presents some significant problems in experimental design. An alternative approach is to measure a chemical whose source is tobacco-specific. In the case of nitrosamines, several tobacco-specific nitrosamines have been identified in mainstream and sidestream smoke[9]. To date, these have not been reported in ETS.

In addition to the possible presence of carcinogens in ETS, it has been suggested that the nitric oxide found in ETS may present a carcinogenic risk by increasing the formation of endogenous carcinogens[10]. Brunnemann _et al._[11]

using the urinary excretion of N-nitrosoproline (NPRO) as a marker for endogenous N-nitrosation of amines, report no increase in the NPRO excretion in individuals experimentally exposed to ETS for three 80 minute periods a day for two days. Excretion of NPRO was greater in smokers than in non-smokers.

Finally, risk extrapolation based on the presence of carcinogens, in the absence of appropriate epidemiological data, does not *establish* causation. It *presumes* causation. Such a presumption in other contexts -- for regulatory purposes, in considering worst-case conditions -- may not be entirely inappropriate. But it is obviously illogical to test the *possibility* of causation by a method that, at the onset, requires the *presumption* of causation.

The final approach to the problem of evaluating what, if any, causative relationship exists between ETS exposure and lung cancer is obvious:

3. Epidemiological studies based on exposure to ETS *per se*.

This is the only approach that deals directly with ETS rather than with presumed effects of ETS that one derives from the study of surrogates.

Epidemiological Studies of Lung Cancer in Non-smokers and ETS

During the past six years, a number of studies have been published that examine the relationship between exposure to ETS and lung cancer in non-smokers. They fall into two categories according to their experimental design: retrospective case-control studies and prospective longitudinal studies. Several studies have not been included in this review. The study of Knoth, *et al.*[12] has been excluded as it includes no control population. The case control study of Sandler and coworkers[13-15] and the record linkage study of Gillis, *et al.*[16], both of which investigated exposure and multiple disease endpoints in smokers and non-smokers, have been excluded because they included too few cases of lung cancer in non-smokers for analysis. Similarly, Miller[17] is not reviewed here as it deals with overall cancer incidence and does not examine lung cancer specifically.

The case-control studies of ETS and lung cancer differ with respect to definition of both cases and the controls. Cases may be defined broadly as all cancers[13-17], lung cancer of all types[18-21] or defined to include or exclude specific forms of lung cancer[22-25]. Depending on the study, cases were ascertained through hospital records[19,20,22], tumor registries[25] or new diagnoses[21,23,24]. Controls likewise vary in definition and in recruitment. Controls were identified as individuals with other cancers[19], patients with orthopedic[18,24] or other[20,22] conditions, relatives or friends[13,15] or residents of the area[21,25]. The criteria for matching cases to controls were again variable from study to study. In most studies, matching was on the basis of age, sex and/or race. One study also matched for socioeconomic factors, including occupation or education[21] or hospital from which the case was identified[19,20,22]. In four of the studies[21-25], cases and controls were not matched for smoking status.

Because of the variability in study design, data presentation and interpretation, it is impossible to summarize the case control studies in a tabular form. As a result, the major features of the design and findings of each study are discussed briefly below. In reviewing each study, special attention has been given to the reliability cf the lung cancer diagnosis and the methods used to assess and quantitate ETS exposure. A finding of increased risk or a dose-response relationship is reported only when it was found to be statistically significant. The pattern of nonstatistically significant elevated risks will be considered later in this paper.

Chan and Fung[18] compared a study population of 84 nonsmoking, Chinese women with a diagnosis of lung cancer with 139 controls. Lung cancer diagnosis was confirmed histologically in the majority of cases. Spousal smoking habits were determined by personal interview with the subjects. On the basis of this interview, subjects were categorized as passively exposed or nonexposed. The criteria for categorization were not defined. The method of ascertaining the smoking status of the subject was also not defined. The odds ratio for ETS exposure was less than 1.

Correa, et al.[22] identified 10 male and 25 female nonsmoking lung cancer cases from admission and pathology records of 29 participating hospitals. All cases of lung cancer were confirmed histologically. Controls were randomly selected from patients in the same hospital who did not have smoking related disease. There is no indication that the authors attempted to confirm the nonsmoking status, nor is the definition of "nonsmoking" given. Exposure to ETS was determined by personal interview with the subject or next of kin and included questions on both spousal and parental smoking. An attempt was made to quantitate exposure by classifying according to pack-years smoked (1-40 or 40+) by the spouse. With respect to spousal smoking, the only statistically significant relationship was among the nonsmoking females whose husbands smoked more than 41 pack-years. The lung cancer incidence classified according to amount smoked does not give clear evidence of a dose response relationship. Thus the likelihood ratio chi-square is nonsignificant, whereas the Mantel--Haenzel chi-square is significant at the 5% level. Note also that the number of female cases (5) with husbands who smoked 1-40 pack years is less than the number expected (6.3) from the distribution of the 133 female controls. The authors also examined the relationship between parental smoking and lung cancer. Although some associations were found between parental smoking and lung cancer incidence in active smokers, no such relationship was demonstrated among nonsmokers.

The case control study of Garfinkel, et al.[19] included 134 cases of lung cancer among non-smokers identified from four hospitals between 1971 and 1981. All lung cancers were verified by independent histologic examination. For both cases and controls, the identification of nonsmoking status was initially made through the hospital chart, but was reliably confirmed through personal cancer case. Exposure to ETS was determined by personal interview based on a standard questionnaire. The respondent was either the subject, spouse, offspring or close friend. Several approaches were used in the estimation of ETS exposure. Spousal smoking, in terms of total cigarettes smoked per day or number of cigarettes smoked in the home was determined. In addition, data were gathered on the average total number of hours per day that the subject was exposed to ETS. Average exposure during the previous five years as well as the previous 25 years was estimated and was further characterized as exposure in the home, workplace or other areas, or during childhood. The odds ratios -- calculated for nonsmoking females exposed to ETS over the last five years, last 25 years, from a husband who smokes, or a husband who smokes at home -- were not statistically significant. When the data were analyzed according to the number of cigarettes per day smoked by the husband, either total or at home, a statistically significant dose-response relationship was demonstrated. However, when the data were analyzed according to the total number of hours/day of ETS exposure, either over the last five years or over the last 25 years, no relationship was found. Exposure to ETS as a result of parental smoking was found not to be a risk factor for lung cancer.

Kabat and Wynder[20] reported on 29 male and 53 female lung cancer cases identified from hospital charts of newly diagnosed lung cancer patients. Nonsmoking status was determined by a review of the hospital chart. There was no indication that this was confirmed during the interview. The hospital records were checked for histological confirmation of lung cancer, but no independent verification was performed. Controls were nonsmoking hospital patients with non-tobacco related diseases. Exposure to ETS was assessed through a personal interview with the subject using a standardized questionnaire. Three schemes were used to categorize ETS exposure: current exposure on a regular basis in the home; current exposure on a regular basis at work; and having a spouse who had ever smoked. Among the male cases, a statistically significant increased risk was seen only for ETS exposure at work. The authors report a statistically significant dose-response relationship between the frequency of exposure and lung cancer incidence in males. However, the data are not provided and frequency of exposure is not defined. Among the female lung cancer cases, no effect of ETS exposure either in the home or at work, was seen.

Koo, et al.[21] reported on 200 lung cancer cases with histological verification matched with 200 controls by age and socioeconomic status. The method of selection of the controls was not specified. When the analysis was limited to non-smokers, the case and control groups numbered 66 and 97, respectively. Environmental tobacco smoke exposure was estimated by means of a personal interview with the subject. The exposure categories included ever smokers, individuals exposed in the home, in the workplace, in both the home and the workplace, or not exposed. Exposure to ETS either in the workplace, home or both had no statistically significant influence on lung cancer incidence. Similarly, when analyzed according to total hours or years of ETS exposure, no dose-response relationship was observed.

Similarly, when analyzed according to total hours or years of ETS exposure, no dose-response relationship was observed.

The study of Trichopolous, *et al.* was published in 1981[23] with an update published in 1983[24]. Since the reports are methodologically identical and differ only with respect to the total number of cases and controls, only the findings of the latter study will be discussed here. Cases consisted of 102 Greek women, ascertained from three hospitals, with a diagnosis of lung cancer other than adenocarcinoma or terminal bronchial carcinoma. For the most part there was no attempt to verify the lung cancer diagnosis by independent histologic examination. Controls consisted of 251 patients identified from a nearby orthopedic hospital. Although not stated, it would appear that controls were matched to cases for age, occupation and schooling. Smokers were not initially excluded from the case and control populations, but were excluded from subsequent analyses. The subsamples of nonsmokers consisted of 77 cases and 225 controls. Ex-smokers, current smokers and nonsmokers were defined, and ETS exposure was estimated based on the number of cigarettes smoked per day by the husband. This information was ascertained from a personal interview with the study subjects. In the paper, the data on lung cancer incidence were tabulated according to ETS exposure and categorized as 1-10, 11-20, 21-30 or 31 or more cigarettes per day smoked by the spouse. An analysis of this table by chi square or the method of Grizzle-Stamer & Koch shows no statistically significant association ($p=0.19$). For reasons which are not explained, odds ratios are calculated for combined exposure groups. That is, the 1-10 and 11-20 groups are combined and the 21-30 and 31+ groups are combined. This analysis yields odds ratios that appear to demonstrate a statistically significant linear trend. However, the odds ratios for each individual exposure group are, respectively, 1.4, 2.6, 4.3 and 2.9, using non-smokers as a baseline. The apparent decline in risk in the highest exposure group suggests that the evidence for a dose-response relationship is equivocal.

The study of Wu, *et al.*[25] was restricted to white females in Los Angeles. Cases consisted of 149 women with adenocarcinoma of the lung and 71 women with squamous cell carcinoma of the lung. All diagnoses were confirmed histologically. Controls were selected from neighbors of the cases and matched one to one by sex and age. Both cases and controls included active smokers. Environmental tobacco smoke exposure was approximated on the basis of a telephone interview using a standardized questionnaire. When the analysis was limited to nonsmoking cases and controls, there were 29 cases of adenocarcinoma and two cases of squamous cell carcinoma. The risk of adenocarcinoma was not affected by exposure to ETS. There were too few cases of squamous cell carcinoma among nonsmokers for analysis.

In addition to the case-control studies, two large-scale longitudinal studies of ETS and lung cancer in non-smokers have been published. Both were non-randomized prospective studies in which two samples of nonsmoking subjects were identified by the presence or absence of an antecedent factor, here, exposure or non-exposure to ETS. These subjects were then followed forward to compare the proportions of the two groups developing lung cancer.

that death certificates were used. However, 93% of these volunteers were followed over 12 years by means of four follow-up questionnaires. Classification of smoking status was by self-report at the beginning of the survey. Very few non-smokers reported that they had started to smoke on the subsequent questionnaires. Person-years of exposure were aggregated into five year groups. A total of 175,000 nonsmoking wives were classified by their husbands' smoking status. In spite of the large numbers followed, no statistically significant increase was found for possible exposure to husbands who reportedly smoked either less or more than 20 cigarettes per day. A synthetic analysis, matching for age, race, educational status, residence, and husband's occupation gave figures which were likewise not statistically significant.

Hirayama[27-28] reports on a longitudinal study begun in 1965 of 143,000 women and 123,000 men aged 40 and over. Record linkage to death certificates show that 200 lung cancer deaths had occurred in the previous 16 years among 90,000 nonsmoking wives for whom husband's smoking status was known. Smoking status was determined at the start of the study by interviewing the husband and wife independently. Only 5% of those approached refused to participate[27]. The standardized mortality ratios (SMR's) for lung cancer in nonsmoking wives were 1.0, 1.4, 1.6 and 1.9 according to the husband's reported smoking status (nonsmoker, ex-smoker, 1-14, 15-19 and 20+ cigarettes/day, respectively). SMR's for nonsmoking husbands were 1.0, 2.1 and 2.3 according to whether the wife was a non-smoker or smoked 1-19 or 20+ cigarettes/day. Only the SMR of 1.9 for nonsmoking wives of husbands who reportedly smoked more than 20 cigarettes/day was statistically significant. A statistically significant relationship between amount smoked by the husband and lung cancer risk was also reported.

Hirayama describes his study as "a prospective cohort study on the health consequences of cigarette smoking" in which "a clear cut dose response relationship was observed"[29]. However this study is not strictly a prospective or a cohort study but a record linkage study with no attempt to monitor subjects annually and with unspecified losses from the original 1965 sample. No consideration was given to a number of variables that could have influenced the outcome of the study, such as the validity of the subject's nonsmoking status and estimates of ETS exposure, the accuracy of the lung cancer diagnosis and the success of matching exposed and non-exposed subjects to account for possible confounding factors.

Discussion

The two types of experimental designs, case-control and longitudinal, used to evaluate the relationship between exposure to ETS and lung cancer in non-smokers, each have advantages as well as limitations. The case-control study is an effective design for suggesting hypotheses but not for confirming them since, by the nature of the design, it is subject to bias. Bias is introduced both in the selection and recruitment of controls as well as in the estimation of exposure to ETS. Controls and cases are generally recruited them since, by the nature of the design, it is subject to bias. Bias is introduced both in the selection and recruitment of controls as well as in the estimation of exposure to ETS. Controls and cases are generally recruited following an explanation of the purpose of the study and may decide whether or not to participate based on their exposure to ETS. If a non-smoking control with no ETS exposure is more likely to participate than one with ETS exposure, the small selection bias in the controls will be reflected in all subsequent odds ratios since this group of controls forms the baseline for such comparisons.

Case-control studies are also subject to respondent bias in answering questions concerning ETS exposure. In the situation, for example, of a non-smoking mother's death, the response from her off-spring regarding the father's smoking habits and mother's exposure is likely to differ according to whether the mother had died from lung cancer or a cause not thought to be related to tobacco. The influence of respondent bias is demonstrated in the case-control study of Garfinkel *et al.*[19]. These authors examined the effect of respondent identity on the odds ratios for exposure categories. When odds ratios for husband's smoking habits were categorized according to respondent identity, the highest odds ratios were seen when the son or daughter was the respondent. In fact, for husband's smoking at home, odds ratios were <1 when the respondent was the subject, husband or close acquaintance, and 3.19 when son or daughter was the respondent.

Longitudinal studies are less subject to selection and respondent bias since the recruitment of subjects and estimation of exposure are done prior to the development of disease. However, longitudinal studies, as well as case-control studies, must be carefully evaluated for the authors' awareness of and correction for possible confounding factors, as well as for systematic misclassification that would affect the experimental outcome.

Among the confounding factors of significance in studies of ETS and lung cancer are age, race, socioeconomic status, marital status, smoking status, ETS exposure outside the home and exposure to chemicals from other sources. In the case-control studies, the cases and controls are matched for a variety of confounding factors. However, in a number of studies[21-24] the matched populations include both smokers and non-smokers. Thus, although cases and controls may have been matched for other confounding variables, when the investigator selects the subgroup of non-smokers, in order to study the association of ETS exposure with lung cancer in nonsmokers, this matching is destroyed. Therefore many of the case-control studies are, in fact, both unmatched and unadjusted for such important variables as age, race and socioeconomic status.

The single most important limitation of the epidemiologic studies on ETS and lung cancer, no doubt resulting in significant misclassification, is the inability to document exposure. In all studies, classification according to ETS exposure was based on responses given to questionnaires and/or during personal interviews. Of the 14 studies examining lung cancer incidence in non-smokers, ten considered only ETS exposure occurring within the household and primarily associated with the smoking habits of the spouse. The remaining four studies included some consideration of exposure outside the home, usually in the workplace.

The reliability of spousal smoking habits as a measure of ETS exposure is very questionable. Friedman *et al.*[30] found that 47% of non-smoking wives of smoking husbands in the United States report no exposure to ETS in the home. Further, 40% of non-smoking women married to non-smokers reported significant ETS exposure outside the home. Thus classification of exposure according to the smoking status of the husband is likely to result in substantial misclassification.

Of the four studies that have considered ETS exposure outside the home, three have attempted to define some composite measure of exposure from several sources[19,21,25]. None of these analyses yielded a significant relationship between composite ETS exposure and lung cancer. The case-control study of Garfinkel, *et al.*[19], which represents the most rigorous effort to date to document total ETS exposure, found no dose-response relationship between the hours/day of ETS exposure from all sources and lung cancer risk.

In addition to ETS exposure, there are two other sources of misclassification common to several of the studies. The first is misclassification of the smoking status of the lung cancer subject. Many of the studies do not describe the method by which non-smoking status was determined, although most studies relied on hospital chart entries and/or interviews. A recent study[19] has shown that hospital charts are an unreliable indicator, with 40% of women reported to be non-smokers actually having smoked at some time during their life. Further, Kabat and Wynder[20] have reported that individuals with smoking-related diseases tend to deny a smoking history more often than controls with no tobacco-related disease. Such differential misclassification of subjects' smoking status in a case-control study would be expected to greatly increase the odds ratio.

Misclassification of smoking status of lung cancer cases in studies could also significantly affect the outcome of longitudinal studies. In any study there will be a certain percentage of smoking respondents who will deny their smoking status and be classified as non-smokers. This percentage may be higher in longitudinal studies than in case-control studies since the much greater size of the study population in a longitudinal study will preclude personal interviews and careful probing of the reliability of responses. As pointed out by Lee[31], since smokers are more likely to be married to other smokers than non-smokers and, therefore, will be categorized as having a greater exposure to ETS from the spouse, the net result will be expressed as an overestimate of the relative risk.

A final source of misclassification is the lung cancer diagnosis. Garfinkel, *et al.*[19] report that 13% of patients identified through hospital records as lung cancer cases did not, in fact, have primary lung cancer. These authors and others[20] have stressed the importance of histologic confirmation of the lung cancer diagnosis.

As a result of the serious questions raised concerning the sources of misclassification and confounding influences, as well as the lack of consistency of association within and among studies, it is generally accepted that causality has not been established[2,32,33]. However, even in the absence of causality, it is appropriate to evaluate the data for the strength of association, if any, which has been established. It must be emphasized, however, that association does not necessarily imply causality.

Nine epidemiologic studies contain a sufficient number of cases for the analysis of the association between ETS exposure and lung cancer in nonsmokers. As discussed above, most are seriously weakened by likelihood of bias, misclassification and confounding influences. Ignoring these limitations, however, the reported findings of the studies can be collated. In most studies, multiple comparisions were made based on differences in sex, source of exposure (home, workplace, total) and/or amount of exposure. In five of these studies[19,20,22,24,27], at least one of the comparisons demonstrated a statistically significant increase in risk associated with ETS exposure. With one exception[18], all of the studies reported at least one comparison suggesting an elevated risk associated with ETS exposure, whether statistically significant or not. Seven studies provided data on lung cancer risk as a function of some estimate of amount of ETS exposure (dose-response). Of these, four[19,20,24,27] reported trends that were statistically significant. However, the dose-response reported by Kabat and Wynder[20] is at this time uninterpretable since the measure used (frequency of exposure) was undefined and no supporting data were given.

In spite of what appears to be a relatively consistent finding among epidemiologic studies of an association between ETS exposure and lung cancer in non-smokers, the specific inconsistencies within as well as among studies must also be considered. The studies of Hirayama[27], Trichopoulous, _et al._[24] and Correa, _et al._[22] present the most consistent data with respect to an association between lung cancer and ETS exposure. These studies each involved lung cancer incidence in women as a function of the smoking habits of the husband. The case-control study of Garfinkel, _et al._[19], while strongly supporting this specific association, could identify no relationship between lung cancer and composite ETS exposure from all sources. The prospective study of Garfinkel[26] reported an elevated risk in nonsmoking women married to smokers. However, the lack of a dose-response relationship coupled with the lack of statistical significance suggest that this study does not support an association between ETS and lung cancer. Of the remaining three studies, one[21] reported a statistically non-significant elevation in risk, but no dose-response relationship and one[20] reported six different comparisons based on sex and source of ETS of which only one was elevated. Thus, any association between ETS and lung cancer can not be considered consistent.

Moreover, in considering the equivocal epidemiological literature that can be and has been subject to differing interpretations, it is important to distinguish between the establishment of an association and causality. The difficulties associated with the documentation of ETS exposure, the inconsistencies within and among studies, and the absence of statistical significance in many of the comparisons all caution against overinterpretation of the data with respect to causality. In order to conclude that ETS is the cause of any elevated risk observed, the following alternative explanations for the association must first be ruled out:

1. Any association may represent sampling error. However, the number of studies reporting odds ratios significantly greater than one speaks against this explanation.

2. An elevated odds ratio may be due to systematic bias and/or misclassification[33,34]. For example, the similarity in these studies in the methods used to ascertain subjects and to elicit information via questionnaires could result in a consistent bias in reporting smoking status and exposure.

3. An elevated odds ratio may be due to the fact that any effect of ETS has been significantly confounded with an unmeasured factor or factors and is merely serving as a marker for this factor.

4. An elevated odds ratio may be due to ETS exposure in combination with other factors, e.g., other indoor air pollutants. Again, this explanation should not be accepted until other "design dependent" explanations have been ruled out. If accepted, the relative importance of each factor, as well as their interactions, should be determined.

5. Two or more of the above possibilities may be operating.

The influence of confounding variables on studies examining the relationship between ETS exposure and lung cancer has received relatively little attention. For example, women married to non-smokers may be more likely than those married to smokers to avoid any environment that is not, to their minds, "clean." Thus they may avoid occupational or environmental exposures to potentially hazardous chemicals. This is but one of numerous potential confounding factors which may influence the outcome of the studies.

The findings of the case-control study of Garfinkel, et al.[19] which is clearly the most rigorous in attempting to avoid misclassification, suggest that, in fact, confounding may play a significant role in studies that use the husband's reported smoking habits as an index of exposure. In this study, a dose-response relationship was suggested between the reported number of cigarettes smoked by the husband (whether total or only at home) and lung cancer. However, no relationship existed between lung cancer and estimated total daily exposure to ETS from all sources including the husband. The authors, in attempting to explain this finding, suggest that classification of ETS exposure according to number of hours of exposure daily is a poor estimate of the intensity of exposure. However, there is no reason to believe that the total daily exposure to ETS from all sources including the husband. The authors, in attempting to explain this finding, suggest that classification of ETS exposure according to number of hours of exposure daily is a poor estimate of the intensity of exposure. However, there is no reason to believe that the husband's smoking habits will provide any better estimate of exposure intensity. One could argue equally well that total hours of exposure, especially over the course of 25 years, is much more likely to reflect lung cancer risk than an isolated source of exposure especially when the estimate of that isolated source does not consider the duration of exposure or other sources of exposure. A more likely explanation for the findings of Garfinkel et al. is that the analysis of lung cancer risk as a function of the husband's reported smoking habits is highly confounded by some unidentified factor or factors. The husband's smoking habits would then be acting as a marker or surrogate for the underlying, as yet unidentified, source.

Conclusions

The epidemiological data concerning any cause and effect relationship between ETS and lung cancer is, at best, equivocal. The most persistent weakness is the absence of adequate exposure information. This precludes the evaluation of dose-response relationships -- a critical element of the test for causality. Moreover, problems of sampling bias and misclassification complicate interpretation of studies done to date.

Acknowledgment

This work was done in association with the Indoor Air Pollution Advisory Group of the Center for Environmental Health and Human Toxicology and was supported by a contract from the Tobacco Institute, Washington, D.C.

REFERENCES

1. M.W. First, "Constituents of sidestream and mainstream tobacco smoke and markers to quanitify expsoure to them," In: Indoor Air and Human Health, Gammage and Kaye, eds. Lewis Publishers Inc., Chelsea, MI, 1984, pp. 195-203.

2. U.S. Department of Health and Human Services, "Involuntary Smoking,"In: The Health Consequences of Smoking. Cancer, A Report of the Surgeon General," 1982, pp. 237-254.

3. C.F. Hiller, K.T. McCusker, M.K. Mazumder, J.D. Wilson, R.C. Bone, "Deposition of sidestream cigarette smoke in the human respiratory tract," Am. Rev. Respir. Dis. 125: 406 (1982).

4. J.D. Brain, B.E. Barry, "Biological potential and exposure-dose relationships for constituents of cigarette smoke," In: Indoor Air and Human Health, Gammage and Kaye, eds. Lewis Publishers, Inc., Chelsea, MI., 1984, pp. 215-225.

5. U.S. Department of Health, Education and Welfare. "Involuntary Smoking," Chapter 11, In: Smoking and Health: A Report of the Surgeon General, 1979, p. 11-6.

6. K.D. Brunnemann, J.D. Adams, D.P.S. Ho, D. Hoffmann, "The influence of tobacco smoke on indoor atmospheres II. Volatile and tobacco specific nitrosamines in main-and sidestream smoke and their contribution to indoor pollution, In: Proc., 4th Joint Conference on Sensing of Environmental Pollutants, American Chemical Society, 1977, pp. 876-880.

7. K.D. Brunnemann, D. Hoffmann, "Chemical studies on tobacco smoke, LIX. Analysis of volatile nitrosamines in tobacco smoke and polluted indoor environments," IARC 19: 343 (1978).

8. G. Stehlik, O. Richter, H. Altmann, "Concentration of dimethylnitrosamine in the air of smoke-filled rooms," Ecotoxicol. Environ. Safety 6: 495 (1982).

9. D. Hoffmann. K.D. Brunneman, J.D. Adams, S.S. Hecht, "Formation and analysis of N-nitrosamines in tobacco products and their endogenous formation in consumers," IARC Sci. Publ. 57: 745 (1984).

10. S.S. Mirvish, "Invivo formation of N-nitroso compounds. Formation from nitrate and nitrogen dioxide, and relation to gastric cancer." In: Nitrosamines and Human Cancer (Banbury Report No. 12), Magee, ed. Cold Spring Harbor, N.Y., 1982, pp. 227-236.

11. K.D. Brunnemann, J.C. Scott, N.J. Haley, D. Hoffmann, "Endogenous formation of N-nitrosoproline upon cigarstte smoke inhalation, " IARC Sci. Publ. 57: 819 (1984).

12. A. Knoth, W. Bohn, F. Schmidt, "Passive smoking as a causal factor for bronchial carcinoma in female non-smokers," Med. Klin. 78: 66 (1983).

13. D.P. Sandler, R.B. Everson, A.J. Wilcox, "Passive smoking in adulthood and cancer risk." Am. J. Epid. 121: 37 (1985).

14. D.P. Sandler, R.B. Everson, A.J. Wilcox, J.P. Browder, "Cancer risk in adulthood from early life exposure to parent's smoking." Amer. J. Publ. Health 75 (2): 487 (1985).

15. D.P. Sandler, A.J. Wilcox, R.B Everson, "Cumulative effects of lifetime passive smoking on cancer risk." Lancet 1: 312 (1985).

16. C.R. Gillis, D.J. Hole, V.M. Hawthorne, P. Boyle "The effect of enviornmental tobacco smoke in two urban communities in the west of Scotland" Eur. J. Resp. Dis. 65 (suppl 133): 121 (1984).

17. G.H. Miller, "Cancer, passive smoking and nonemployed and employed wives." West. J. Med. 140: 632 (1984).

18. W.C. Chan, S.C. Fung, "Lung Cancer in non-smokers in Hong Kong. "In: Cancer Campaign, Vol 16: Cancer Epidemiology, Grundmann, ed., Gustav Fisher Verlag, Stuttgart, 1982, pp. 199-202.

19. L. Garfinkel, O. Auerbach, L. Joubert,"Involuntary smoking and lung cancer: A case control study." J. Natl. Canc. Inst. 75(5): 463 (1985).

20. G.C. Kabat, B.L. Wynder, "Lung cancer in non-smokers." Cancer 53: 1214 (1984).

21. L.C. Koo, J.H.C. Ho, D. Saw, "Is passive smoking an added risk factor for lung cancer in Chinese women." J. Exp. Clin. Cancer Res. 3(3): 277 (1984).

22. P. Correa, L.W. Pickle, E. Fontham, Y. Lin, W. Haenszel,"Passive smoking and lung cancer." Lancet 2: 595 (1983).

23. D. Trichopoulos, A. Kalandidi, L. Sparros, B. MacMahon, "Lung cancer and passive smoking." Int. J. Cancer 27: 1 (1981).

24. D. Trichopoulos, A. Kalandidi, L. Sparros, "Lung cancer and passive smoking: conclusions of the Greek study." Lancet 2: 677 (1983).

25. A.H. Wu, B.E. Henderson, M.C. Pike, M.C. Yu. "Smoking and other risk factors for lung cancer in women". J. Natl. Canc. Inst. 74(4): 747 (1985).

26. L. Garfinkel "Time trends in lung cancer mortality amontg non-smokers and a note on passive smoking." J. Natl. Canc. Inst. 66(6): 1061 (1981).

27. T. Hirayama,"Non-smoking wives of heavy smokers have a higher risk of lung cancer: A study from Japan." British Medical Journal. 282: 183(1981).

28. T. Hirayama, "Cancer mortality in nonsmoking women with smoking husbands based on a large scale cohort study in Japan". Prev. Med. 13:680 (1984).

29. T. Hirayama, "Letter to the editor," Brit. Med. J. 282: 1393 (1981).

30. G.D. Friedman, D.B. Petitti, R.D. Bawol, "Prevalence and correlates of passive smoking," Amer. J. Publ. Health 73: 401 (1983).

31. P. Lee. "Round table discussion." From the symposium "Medical Perspectives on Passive Smoking," Prev. Med. 13: 733 (1984).

32. S.T. Weiss, "Passive smoking and lung cancer. "What is the risk?":, Amer. Rev. Resp. Dis. 133:1 (1986).

33. J.M. Samet, "Relationship between passive exposure to cigarette smoke and cancer, " In: Indoor Air and Human Health, Gammage and Kaye, eds. Lewis Publishers, Inc., Chelsea, MI, 1984, pp. 227-240.

34. V. Gladen, W.J. Royan, "Misclassification and the design of environmental studies," Am. J. Epidemiol. 109:607 (1979).

PENNSYLVANIA
DER

COMMONWEALTH OF PENNSYLVANIA
DEPARTMENT OF ENVIRONMENTAL RESOURCES

TESTIMONY FOR THE RECORD OF SEPTEMBER 17, 1986

HEARING ON RADON AND INDOOR AIR QUALITY RESEARCH

HOUSE COMMITTEE ON SCIENCE AND TECHNOLOGY SUBCOMMITTEE ON NATURAL RESOURCES, AGRICULTURE RESEARCH AND ENVIRONMENT

The Pennsylvania Department of Environmental Resources (DER) appreciates the opportunity to update the Subcommittee on activities in Pennsylvania since Nicholas DeBenedictis, Secretary of DER, testified before this Subcommittee on October 10, 1985.

Radon is not an issue Pennsylvania chose, but it is a responsibility that, once discovered, we have accepted and vigorously pursued in order to safeguard the health of our citizens. Since the discovery in December 1984 of a home with levels 675 times the exposure level generally deemed safe, Pennsylvania has led the nation in testing, monitoring and implementing remedial designs to correct radon problems in homes. By the end of our fiscal year 1986-87, the Commonwealth of Pennsylvania will have committed $6.4 million in detection, research and mitigation measures. We are the only state offering extensive free radon testing, the first to offer affordable loans to homeowners and the first to undertake its own remediation demonstration program.

We now know that radon is a problem not just in Pennsylvania, but of national, even international, scope. We are also beginning to understand that when exposure is high, the risks to human health may be of an order of magnitude eclipsing many other environmental risks. In recognition of this, for the past 18 months or so, the federal government has been working with the Commonwealth in a way we hope is becoming a model for federal/state relations. We need this relationship to continue and to expand. We commend the many Pennsylvania cosponsors for introducing legislation to better define EPA's continuing role in radon mitigation research and development.

An effective national radon action program must be a federal, state, local and private partnership. There are several areas in which continuing federal research will be necessary to complement our state's actions:

- although a preliminary analysis of lung cancer mortality rates for selected areas affected by high residential radon exposure in Pennsylvania was conducted, nationwide epidemiological studies will be necessary to refine risk assessments, since existing studies have been based on occupational exposure standards;
- continuing development and evaluation of remedial techniques in varying situations will expand the data base for all states. In particular, it may be more cost-effective to undertake research at the federal level to develop built-in remedies for new home construction in radon-prone areas;
- refinement and application of methods to better predict the location of radon-prone areas.

All research needs to be coordinated with the federal/state action programs to ensure that research dollars are being spent effectively and where they are most needed.

PENNSYLVANIA'S INITIATIVES

Most of Pennsylvania's activities to date have focused on the Reading Prong, a geological formation running through four Pennsylvania counties as well as three other states.

Detection

DER has offered free radon screening detection kits in the Pennsylvania portion of the Reading Prong with over 21,000 kits sent to date. Of the over 17,000 homeowner test results to date, approximately 60% of them have been found to have radon concentrations over .02 working levels. In addition, we have sampled every public school building - 131 in all - in the Reading Prong. The radon levels in these buildings appear to be less than residences, both in terms of proportion of buildings exceeding the action level and levels of the highest readings.

Outside the Reading Prong, we have been directing residents to reputable sources of radon detection kits, so that homeowners will not be taken advantage of by unscrupulous individuals.

DER is planning to extend its radon survey to isolated pockets in eastern Pennsylvania which demonstrate the potential for radon exposure. Our geologists are now attempting to prioritize new survey areas of potential risk.

Evaluation and Remediation Demonstrations

Last year, EPA conducted a remediation demonstration program on 18 homes in the Reading Prong; an additional number of homes are to be done in the next year. On May 16, 1986, Pennsylvania Gov. Dick Thornburgh signed into law a $1 million state program to develop, install and evaluate methods to reduce unsafe radon levels in homes. DER is now in the process of selecting contractors and developing the criteria to select homes for this program. This program will not only provide participating homeowners with installed remediation systems but is also designed to increase our knowledge of radon reduction techniques so that future efforts can be efficient, effective and readily transferrable to other similar situations.

Though we recognize that much still needs to be done to evaluate remediation techniques, it is essential that residents of homes with high levels of radon be able to reduce their exposure. Therefore, Pennsylvania has also made available a low interest loan program for eligible homeowners with radon levels above .02 WL. DER is assisting the Pennsylvania Housing Finance Agency in administering this program, as well as establishing material and work standards for both the loan and remediation programs.

One of the most important elements in establishing an effective remediation program is ensuring the availability of properly qualified contractors and consultants. To that end, DER has sponsored free courses for home improvement professionals during July and August 1986. Almost 100 people representing 65 companies and organizations participated. The courses were developed by EPA, and we hope that they can be presented again in Pennsylvania.

Health Studies

The Pennsylvania Department of Health conducted a lung cancer mortality study around the area where high levels of radon were originally detected. The rate of cancer deaths was found to be consistent with the rest of the state. This year, PA began working with Argonne National Laboratories on a more comprehensive 5-year cancer study under the direction of the U.S. Department of Energy.

Public Education and Participation

DER has developed education materials which indicate the health risks of radon, and describe measures which could be taken to reduce radon levels in homes. DER is also making available two recently produced EPA pamphlets which provide basic information on radon risks, detection and remediation.

A citizens committee was recently appointed to advise DER on radon issues. The Committee, which held its first meeting on September 30, will give DER guidance in formulating future radon research and mitigation actions.

In conclusion, we are continuing our aggressive program to protect our citizens from the serious health threat posed by radon. We appreciate the Subcommittee's continuing interest in this area and Congress' desire to ensure an appropriate level of federal support for radon programs.

RADON INFORMATION COUNCIL
1250 Eye Street, N.W. Suite 300 Washington, D.C. 20005
(202) 682-0690

STATEMENT OF PURPOSE

The Radon Information Council is a national organization of industry associations, corporations and professionals involved in all phases of construction and home building, and others involved with the science of the issue.

The Radon Information Council was established in April, 1986, to keep industry and the media informed about radon testing, prevention and mitigation. In addition, the Council is working with the public and private sectors to assure that the public receives accurate and responsible information about the issue of radon gas in the home.

Members of the Council include:

- National Concrete Masonry Association
- National Association of Home Builders
- National Ready Mixed Concrete Association
- National Society of Professional Engineers
- Expanded Shale, Clay & Slate Institute
- Portland Cement Association
- National Stone Association
- Home Owners Warranty Corporation
- Terradynamics Corporation

The Radon Information Council is managed by a steering committee made up of representatives from each of the member organizations. In addition, four other committees -- technical, liability, legislative/regulatory and public information -- handle issues in those areas.

The Council is chaired by John Heslip, president of the National Concrete Masonry Association. Martin Mintz of the National Association of Home Builders serves as vice-chair.

# # #

080186

RADON INFORMATION COUNCIL
1250 Eye Street, N.W. Suite 300 Washington, D.C. 20005
(202) 682-0690

Testimony of
John A. Heslip, Chairman
Radon Information Council

House Committee on Science & Technology
Subcommittee on
Natural Resources, Agriculture Research & Environment
September 17, 1986

Mr. Chairman, I commend the committee for calling this afternoon's hearing. The topic of indoor air quality is extremely complex, and it seems the more we learn about it, the more complex it becomes.

I come before the committee today to discuss one particular indoor air contaminant -- radon.

The Radon Information Council was formed earlier this year by representatives of industry who were worried that in all the attention being paid to the demon radon we were neglecting to look at what can easily be done to limit or eliminate the problem.

While we have known about the potential health hazard of radon gas for many years, it has only been in the last few that we have turned our attention to its presence in the home; and the danger posed by it.

By now I am sure you all know the story of Stanley Watras and his home in Boyertown, Pennsylvania. The media never fail to cite it when doing a story on radon. So too do policy makers when discussing the need to somehow control radon by regulation.

As more and more media attention is paid to radon, I can safely predict that more and more pressure will be put on policy makers such as yourselves to somehow regulate radon out of our lives. If it was that easy I would be at the head of the line in the call for such action.

However, reality is seldom so simple, and I suggest to the committee that we need to learn much more about radon before we can intelligently move to lessen it as a problem.

A quick look at sensational media coverage of radon points out how much misinformation is reaching the public. As a representative sample I give you the following headlines:

- "Radon: New Cancer Threat," Good Housekeeping, 2/86.
- "Radon Called Biggest Threat to Health of Americans," Journal of Commerce, 3/4/86.
- "Risk of Cancer From Radon Gas Increases With Growth of Energy-Efficient Homes," Wall Street Journal, 2/23/84.
- "Hidden In Your Shower: A Cause for Lung Cancer," USA Today, 8/5/86.

- "Your Home Could be Turning Into a Possibly Lethal Gas Chamber," WJLA-TV News, 2/11/86.

- "Your House May Be a Death Trap," U.S. News and World Report, 3/17/86.

To set the record straight, radon is not a new cancer threat. Scientists and physicians long ago linked lung cancer to prolonged exposure to extreme levels of radon.

Nor is radon the biggest health threat to Americans. While it is believed radon is the second leading cause of lung cancer deaths, it is important to keep this in perspective. The U. S. Environmental Protection Agency estimates that radon causes about 9,000 deaths per year, though the number could be as high as 20,000. The media, however, always seem to quote the higher number, without ever saying the total is probably much lower. Cigarette smoking, on the other hand, leads to more than 110,000 lung cancer deaths annually, according to the American Lung Association.

Contrary to what has been reported, the risk of radon exposure does not increase in newer, energy-efficient homes. While that was an early theory, studies conducted by the Bonneville Power Administration on the West coast have found no real increase in radon levels in energy-efficient homes.

And radon does not "hide" in anyone's shower. Studies have shown high levels of radon in some private well water supplies in New England and Rocky Mountain states, but this is no reason for anyone to fear taking a shower. I suspect people ought to be more concerned with slipping in the shower than washing in it.

Needless to say, nobody's home is a "death trap" nor a "lethal gas chamber."

While it is important for the media to alert the public to potentially dangerous situations in their lives, it is also incumbent on them to do so in an intelligent, responsible and accurate fashion. The only benefit to declaring a home to be a "lethal gas chamber" is to increase ratings. It needlessly frightens individuals and may very well cause them to take inappropriate action.

We know that the Congress is being pressured by the public to take some sort of action on radon. But in light of the kind of information, or perhaps more accurately, lack of information, reaching the public about radon gas, the Radon Information Council believes it is important to proceed with care when dealing with radon.

It is apparent that the public knows very little about radon: where it comes from, how it gets into the home, how to detect it, how to eliminate it, what kind of hazard it poses to our health. This ignorance is also evident among local health and housing officials, as well as officials at the state level. Homebuilders, remodelers, contractors, engineers, ventilation experts, realtors -- all are asking for help in understanding radon. Virtually everyday we receive calls from homeowners, builders, state officials, asking for information, answers to their questions, guidance in what to to.

Yet amidst this environment of ignorance we hear calls to legislate, regulate and litigate.

I don't believe radon is an insurmountable problem. While it is unlike anything we have faced before -- it is not a man-made disaster -- we still have the resources and the capacity to control the threat of radon in our homes.

But first we must understand radon itself: how to accurately test for it in the soil, in water supplies and in the home. What constitutes a good testing procedure, and what is a bad one? How often should one test?

We must understand its affect on our health. We know it causes lung cancer, but much of the information about it has been extrapolated from data on lung cancer among workers in uranium mines. Levels of exposure and length of exposure in the home in many respects are still areas requiring further research.

We must better understand how radon enters the home. We know of one instance where a great deal of repair work was done on a home where high levels of radon were detected, only to find the levels did not go down at all. After further testing was conducted researchers determined that the radon was not infiltrating the home through cracks in the basement, or through sump openings or drains, or any of the other traditional means of entry, but rather, through the water supply. Yet all the work done on the house was to seal the other sources of entry. Short of shutting off or filtering the water, nothing could prevent the entry of radon into this particular home.

There is an urgent need to develop ways to mitigate radon in the home. Some are being tested right now. Some methods show a great deal of promise. However, radon is a tricky problem, and one technique used successfully in one home could worsen the problem in another.

All these questions lead to the conclusion that we need to know more. Policy makers are being asked to make policy but have little information with which to work. That is why the Radon Information Council endorses the direction the EPA, and the U.S. Department of Energy have taken in regard to radon. We believe it is a tremendous mistake to set standards and establish rules with only a portion of the information needed to formulate an adequate answer.

Instead, we think it is much more prudent to continue on the present course, albeit at a quickened pace, to complete the research and educate the public. The EPA programs discussed at today's hearing are well intentioned, and should show positive results. So, too, are the programs at the Department of Energy. However, I would suggest to the committee that there is a need to strengthen the cooperation between the federal agencies and the states, who are crying out for information and direction. And equally important there is a need to recruit the expertise and resources of industry, and improve communication.

As a representative of industry, the Radon Information Council has launched an aggressive campaign to educate industry and the media. At the same time, we know that industry has much to offer in helping to solve the radon riddle. We encourage Congress, the federal agencies and the states to take advantage of industry's resources and expertise, and we urge legislators and regulators at all levels not to act just for the sake of telling their respective constituencies that they are doing something. Rather, we advise prudent action, with the full knowledge that radon is not a problem that has arisen overnight, nor is it a problem we can get rid of overnight. We hope you will act with speed, but not with haste. Thank you.

# # #

ACVA Atlantic Incorporated

10378 B Democracy Lane
Fairfax, VA 22030
703 352 0102

September 30, 1986

The Honorable James H. Scheuer
Chairman, Subcommittee on Natural Resources,
Agriculture Research & Environment
Committee on Science and Technology
U.S. House of Representatives
Room 388, HOB Annex 2
2nd & D Streets, S.W.
Washington, D.C. 20515

Dear Mr. Chairman:

I note that your subcommittee recently completed hearings on the status of the government's indoor air quality research program. Among the witnesses appearing was Dr. Jan Stolwijk of Yale University, who noted in his testimony that "one of the most important ways in which indoor air quality problems develop and get solved is through the ventilation systems which serve the buildings."

My own experiences analyzing building ventilation systems for clients such as the federal government, major hospitals, and corporations, corroborates Dr. Stolwijk's observation. I described those experiences last June 27, in testimony before the House Committee on Energy and Commerce.

Enclosed is a copy of that statement. I hereby request that you include it, along with a study conducted by the National Institutes for Occupational Safety and Health with similar findings, in the record of the September 17 hearing.

Thank you.

Sincerely,

Gray Robertson

GR:dh
Enclosures

STATEMENT OF GRAY ROBERTSON
PRESIDENT, ACVA ATLANTIC, INC.
BEFORE THE UNITED STATES HOUSE OF REPRESENTATIVES, COMMITTEE ON ENERGY AND COMMERCE, CONCERNING THE CONTRIBUTION OF ENVIRONMENTAL TOBACCO SMOKE TO INDOOR AIR POLLUTION

INTRODUCTION

I am President of ACVA Atlantic, Inc., a company that specializes in the study and assessment of indoor air pollution. I received a Bachelor of Science Degree with honors in Chemistry and Botany from London University (Queen Elizabeth College) in 1964. Prior to entering London University, I was employed as a bacteriologist with Evans Medical Company Limited, (part of the Glaxo Group of companies) in Liverpool, England, and studied for a Higher Certificate of Education in Botany and Zoology at Liverpool Technical College.

After extensive experience with the Johnson Matthey Company in England, from 1964 through 1973 as a chemist primarily involved in the use of precious metal catalysts in the oil, gas and chemical industries, I became head of that company's chemical division in the United States for the next five years. I then became General Manager of Phosyn International Limited, a company specializing in the development of trace element technology and the manufacture of speciality chemicals. In 1980, I became Director of Winton Laboratories Limited, an English company specializing in the study of indoor air pollution throughout buildings in Europe, Asia and Scandanavia. While at Winton Laboratories, I personally was involved in the study of a number of buildings and in designing and developing methodology to identify the sources of indoor air pollution. I have been licensed to market in this country the technology developed by Winton Laboratories to assess and resolve indoor pollution problems. Pursuant to that license, Peter Binnie (Technical Director of Winton Laboratories) and I incorporated ACVA in the United States in 1981.

ACVA is often called upon to investigate what has become known as "sick building syndrome" in large commercial office buildings. ACVA seeks to identify the causes of indoor air quality problems, and to recommend remedial steps. Over the past five years, ACVA has studied the air quality in 125 major office buildings with over 27 million square feet of space occupied by over 135 thousand people. Clients include major banks, insurance companies, property developers, hospitals, colleges and government agencies. ACVA has tested the indoor air quality in the Department of Health and Human Services, the Social Security Administration, the Longworth Congressional Building, the United States Supreme Court, GSA Regional Headquarters, the Customs and Excise Building and the Coast Guard Building.

H.R. 4488 and H.R. 4546 suffer from the fundamental misconceptions that "tobacco smoke is a major contributor to indoor air pollution," and that "tobacco smoke is the greatest source of . . . harmful . . . pollution" violating particulate matter air quality standards. My building studies, and research conducted by NIOSH,[1] do not support these assumptions. We frequently investigate buildings as a result of complaints from occupants with symptoms such as eye and nose irritation, fatigue, coughing, rhinitis, nausea, headaches, sore throats and general respiratory problems. Our clients often assume that these symptoms are due to environmental tobacco smoke (ETS). However, identical symptoms are found in individuals exposed to formaldehyde, carbon monoxide, oxides of nitrogen, and ozone. Moreover, similar symptoms are reported by individuals with allergies to Aspergillus, Cladosporium, and Penicillium species of fungi, among others, as well as other miscellaneous bacterial aerosols. Exposure to household dusts, cotton fibers, and fiberglass fragments results in many identical symptoms.

This statement will describe some of ACVA's experience with indoor air pollution. ACVA's research bears directly upon the nature and extent of exposure to environmental tobacco smoke experienced by office workers in commercial buildings. ACVA's primary finding -- that environmental tobacco smoke rarely is the cause of the indoor air pollution

---

1/ Melius, James; Wallingford, Kenneth; Keenlyside, Richard; and Carpenter, James; "Indoor Air Quality - The NIOSH Experience," Ann. Am. Conf. Gov. Ind Hyg., Vol. 10 (1984).

problems found in these buildings -- it is important to this Committee's work and contrary to one of the primary findings supporting each of these proposed bills.

ACVA's EXPERIENCE

Over the past five years, ACVA has been called upon to investigate buildings due to air quality complaints from employees, for hygienic reasons, such as to investigate visible dirt in or around heating, ventilating and air conditioning (HVAC) grilles; for preventive maintenance purposes and sometimes as a matter of general interest. On many occasions, initial complaints are thought to be related to the presence of ETS, when symptoms in office workers such as sore and watery eyes, headaches, nausea, coughing and general respiratory problems are reported. While sensitive individuals may exhibit many of these symptoms upon being exposed to high concentrations of tobacco smoke, ACVA seeks to determine the actual nature and origin of such complaints. We have found that it can be misleading to focus only upon the symptoms described by building occupants in attempting to determine the actual cause of poor indoor air quality. Through extensive on-site testing, ACVA seeks to discover the source of the problem and to suggest remedial steps specific to a particular contaminant and situation.

A. Sick Building Syndrome

1. The Impact of Energy Conservation

The complex of symptoms that I already have mentioned has been termed the "sick building syndrome," a condition often thought to result from energy conservation efforts to seal buildings and reduce the infiltration/exfiltration of air. Stringent energy conservation efforts by building managers have reduced the natural infiltration of fresh air that previously existed in many buildings. These steps have exacerbated the often undiscovered problem of a poorly designed and maintained HVAC system.

Without doubt, the desire to reduce operating expenses has been the major obstacle to increasing ventilation rates. Most companies have incorporated energy management programs and new operating budgets based on saving every energy dollar possible. In fact, in my address last year to the Association of Professional Energy Managers, I pointed out that, since their very salaries and bonuses are dependent on reduced costs, it would be an anathema for them to consider increasing energy usage and operating costs by increasing ventilation.

However, forward thinking companies should look beyond the budget constraints of energy managers. Consider the following facts: The average heating, ventilating and air conditioning operating costs of a typical 100,000 square foot building in this area would be $50,000 per annum. A commendable target for energy saving by reducing ventilation may be 25%, resulting in $12,500 per annum.

Now consider the payroll costs for people in that building. A very conservative value based on national averages would be $10,000,000 per annum per 100,000 square feet. Thus each 1% of absenteeism costs $100,000 per annum. Typical absentee rates run at 3-7%, and no less than 50% of all absenteeism is estimated to be due to upper respiratory problems. How many of these absences are due to dusts, bacteria, fungi, fibers, chemicals, ETS, carbon monoxide, oxides of nitrogen; i.e., how many are due to the internal pollution complex?

In short, what does it profit a company to realize $12,500 in energy savings if that small saving potentially causes hundreds of thousands of dollars in absenteeism, not to mention lost worker efficiency. It is not surprising that some European countries, including Denmark, West Germany and Switzerland, have introduced legislation mandating that steps be taken to prevent the build-up of internal pollutants. This country is destined to follow that course, either by slow evolution or legislation, and it may result from court actions brought by individuals or trade unions. Such suits could seek that building owners, designers and operators accept responsibility for the health and welfare of their staff or tenants.

In addition to tightening buildings and sealing windows, building managers have shut down air conditioning systems at night and on weekends in an effort to lower energy costs. When the air conditioning is shut down in humid climates, condensation builds up and settles inside the duct work. If dirt is present in damp duct work, spores and microbes can flourish, only to spread throughout the building once the HVAC system is turned on the next morning. This often results in Monday morning complaints of building odors or building sickness that disappear during the week, only to reoccur the following Monday morning. To save more energy, automatic temperature controllers are used to cycle fans on and off during the day. Vibrations from the start-up of these fans can cause dirt and germs trapped inside ductwork to be dislodged and carried into occupied areas.

Another energy conservation effort that may contribute to sick building syndrome is the recirculation of indoor air, at the expense of fresh outdoor air. This may be a deliberate policy or it may be a result of the designer's shortsightedness. This results in the continuous redistribution of infectious microbes, allergenic dusts and spores from office to office and floor to floor.

Another cause of sick building syndrome is the installation of variable air volume (VAV) air systems that sometimes permit the introduction of contaminated air into an occupied space. For example, we have found that the mixing boxes of some VAV systems are equipped with louvers that open into the ceiling voids in which they are mounted. When specific temperature conditions exist, the louvers are opened and return/exhaust air can be introduced from the void and distributed throughout the system. This operation allows air from the building void, which is often dirty and contaminated, to return to the building space without treatment by the system's filtration devices. Fiberglass particles sometimes are introduced into a building space from this source. During our studies from June 1984 through July 1985, three outbreaks of dermatitis reactions in employees were traced to the introduction of fiberglass particles through VAV mixing boxes. In May 1985, ACVA traced a sickness outbreak in a federal building to the spread of allergenic fungi from its source in the ceiling void through a VAV heater box. In addition, the substitution of low cost, low efficiency filters to reduce pressure drops and save energy seriously reduces the efficiency of building filtration systems, and can lead to serious indoor air quality problems (filters may be not only poor, but poorly installed).

2. Sources of Indoor Air Contamination

Virtually every indoor decoration, building material, or piece of furniture sheds some type of gaseous or particulate pollutant. When a building is new, some compounds are given off quickly and soon disappear. Others continue "outgassing" at a slow pace for years. Clothing, furnishings, draperies and carpets contribute fibers and other particulates. The use of cleaning solvents, detergents and pest control agents, such as chlordane, also may cause persistent problems in office environments. Sweeping, vacuuming and dusting normally remove the larger particles, but often increase airborne concentrations of smaller particulate matter. Common office supplies have been found to release dangerous chemicals, such as formaldehyde (from paper), into the air. Evaluations by NIOSH of indoor office spaces have discovered methyl alcohol from spirit duplicators and methacrylate and ozone from copiers.[2]

Environmental tobacco smoke often is blamed for the symptoms associated with "sick building syndrome." The reason is obvious - environmental tobacco smoke often is the only visible indoor air pollutant. ACVA has determined, however, that the presence of high concentrations of tobacco smoke indicates a much more serious problem of poorly designed and improperly maintained ventilation systems. In this case, ETS, as well as a number of other more significant indoor contaminants, are permitted to accumulate in the indoor space, and air quality deteriorates rapidly.

---

[2] "Indoor Air Quality - The NIOSH Experience," supra at 4.

High levels of environmental tobacco smoke have been determined to be the immediate cause of indoor air problems in only five, or 4% of the 125 major buildings investigated by ACVA since 1981. In a similar study of 203 buildings from 1978-1983, NIOSH found that only 4 of the buildings studied (2%) had indoor air quality problems attributable to high concentrations of ETS.[3] Significantly, where high accumulations of ETS have been found, ACVA has discovered an excess of fungi and bacteria in the HVAC system. These microorganisms usually are found to be the primary causes of the complaints and acute adverse health effects reported by building occupants.

ACVA determines the extent of bacterial and fungal contamination of a building through diverse microbiological sampling techniques, classical air sampling in rooms, air sampling from ducts and surface sampling. While we recognize that such sampling may sometimes be difficult to interpret quantitatively due to factors such as surface irregularities, the discovery of significant concentrations of specific fungi and bacteria is significant. An excess number of these microbial contaminants may be a precursor to future health problems.

---

[3] "Indoor Air Quality - The NIOSH Experience," supra at 4; see also Wallingford, K.M.; "NIOSH Indoor Air Quality Investigations: 1971 through 1985," (repesenting more than 350 building investigations).

Among the bacteria that we frequently have isolated from the HVAC systems of offices are: Staphylococcus aureus, Staphylococcus epidermidis, Coliform species, Diphtheroid bacilli, Pseudomonas species, Proteus species, Bacillus species and Serratia species. Eleven, or 9% of the buildings studied by ACVA have exhibited high levels of potentially pathogenic or allergy causing bacteria. ACVA has found over 27 different species of fungus contaminating air handling systems. The most common isolates include Penicillin species, Mucor species, Aspergillus species, Alternatia species, Rhizopus species, Rhodotorola species, Candida species, Cladosporium species, Fusarium species and Cephalosporium species. Of the over 125 buildings studied by ACVA since 1981, 39, or 31%, have been found to contain high levels of potentially pathogenic or allergy causing fungi. In several buildings with excessive staff complaints, either Aspergillus species and/or Cladosporium species of fungus were found. In some instances, colony counts of airborne fungi have run into the thousands per cubic meter of air. Epidemiological tests run by various doctors have confirmed severe allergic reactions to the spores of these fungi in all affected staff. Subsequent cleaning and removal of the sources of these fungal contaminants have resulted in a complete abatement of complaints. In a similar situation, microbiologist Phil Morey, formerly of NIOSH and now associated with Honeywell, found fungi in a government building in Washington with levels of up to 83,700 fungi per cubic meter, characterizing the building's environment as "sort of like a chicken coop."[4]

Dangerous amounts of fungi and bacteria were discovered in each of the five buildings found by ACVA to contain high levels of environmental tobacco smoke. In each of these buildings, once the HVAC systems were cleaned and ventilation rates were modified, the indoor air quality improved dramatically. Tobacco smoking continued to be permitted throughout the building without any further discomfort or adverse effects being reported.

HVAC systems also have been found to be poorly designed and negligently maintained. Excessive dirt accumulations are common in duct work, even in hospitals. Following the inspection of a number of buildings, hundreds of pounds of fungi, dust and dirt have been removed from the ductwork. Dead birds, insects, rodent carcasses and feces and excess amounts of dust have been found in many buildings where employees have complained of eye irritation, headaches, allergies and general respiratory problems. In addition,

---

[4] "Air Pollution in the Great Indoors," State Legislatures, July/August 1985, at 35.

ACVA often discovers that the fresh air dampers of a building's HVAC system have been closed completely in an effort to save energy. During the summer of 1985, the fresh air dampers were closed completely in over 35% of those buildings studied by ACVA. One misguided engineer actually had bricked up the fresh air vents to save energy. All of these buildings were operating with 100% recycled indoor air. The lack of an adequate fresh air supply, coupled with dangerously low ventilation rates, has led to hazardous ventilation conditions in many of the buildings evaluated by ACVA. Fifty percent of the investigations conducted by NIOSH from 1971-1985 attributed the indoor air quality problems to inadequate ventilation.[5/]

The American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) has suggested a minimum ventilation rate of 15 cubic feet per minute (cfm) per person, but recommended a rate of 15-25 cfm/person in general office spaces (ASHRAE Standard 62-73). As a result of the move to conserve energy in the mid-1970's, ASHRAE sought to revise its existing Standard Natural and Mechanical Ventilation with a new standard describing "Ventilation for Acceptable Indoor Air Quality." (ASHRAE 62-1981). An internal

---

[5/] "NIOSH Indoor Air Quality Investigations: 1971 through 1985," supra at 4.

ASHRAE Revision Committee is about to release a revised version of ASHRAE Standard 62-1981 incorporating a ventilation rate of 20 cfm/person for general office spaces. This is a direct recognition of the potential build-up and accumulation of internal air contaminants permitted by low ventilation rates. The newly revised ASHRAE standard for ventilation is expected to be released for public comment during the summer of 1986; it contains higher ventilation rates for all indoor spaces, an acknowledgment that indoor air pollution problems often can be corrected simply through improved ventilation and increased air movement.

The newly revised ASHRAE standard also will incorporate a minimum recommended ventilation rate of 15 cfm/person when no other rate is specified. This rate was adopted based primarily upon the need to reduce measurable levels of CO_2 to 1,000 ppm. Japan recently adopted this level of acceptable CO_2 concentration, and Canada is seriously considering adopting a rate of 600 ppm as a safety factor. Not surprisingly, a majority of the buildings investigated by ACVA have had measurable ventilation rates far below 20 or even 15 cfm/person. In fact, on many occasions, ACVA has found ventilation rates of 5 cfm/person or lower.

ACVA also conducts measurements to determine the extent of airborne particulate contamination within a building. ACVA routinely measures airborne particulates with electronic light scattering equipment as well as mass particle weight balances. ACVA has found that a major source of high indoor particulate levels is the introduction of outside air into an indoor space. Frequently, the air is unfiltered, partially filtered, or filtered through grossly inefficient filters; as a result, dusts and dirt accumulate in the air conditioning ductwork. Another source of contamination is the construction dirt and debris that accumulates in the ductwork during building construction. In fact, the largest single source of airborne contamination in many buildings is this excessive dirt accumulation inside the air ducts.

Increased human activity also can produce high levels of measurable airborne particulates. Movement within an enclosed space can stir up paper dusts, textile fibers and loose fiberglass particles. Dermatitis due to fibrous glass insulation has been a common problem, usually after the fibrous glass insulation has been disturbed during construction activity. Seven of the 125 buildings (5.6%) evaluated by ACVA since 1981 were found to have heavy concentrations of fiberglass particles in the air.

Recently, during a study of a large bank, ACVA discovered a filtration efficiency of approximately 10% at a particle size of 1.0 micron and larger. The average efficiency of a filtration system in similar buildings ranges from 40 to 50% at this particle size, whereas knowledgeable and concerned building operators can achieve filtration efficiency in excess of 80% at this particle range. The simple replacement of grossly inefficient filters with adequate filters, followed by careful maintenance, often results in a dramatic reduction of airborne particulates. Tobacco smoke here acts as the signal for the existence of a more significant indoor air quality problem, primarily poor ventilation, the lack of an efficient exhaust system and an inadequate supply of fresh air. An overemphasis upon the assumed particle contribution from environmental tobacco smoke could lead a building manager to overlook the much more fundamental problem of a poorly designed and maintained HVAC system. In the five instances in which we have observed high concentrations of tobacco smoke, the smoke accumulation led the building operator to contact ACVA, at which time an in-depth investigation revealed the presence of allergenic fungi that later was discovered to be a major cause of the problems experienced by the occupants.

A novel source of air contamination is related to the presence of an indoor parking garage in the basement of a building. High levels of carbon monoxide in offices have been traced to the exhaust from automobiles in parking garages below the building. This gas can permeate a building through elevator shafts or stairwells. ACVA also has discovered high amounts of carbon dioxide in many buildings, as well as measurable levels of ozone and formaldehyde. Organic compounds are released into the air by building materials, adhesives, aerosols, paint, cosmetics, cleaning solvents, pesticides and burning fuels.

Some of the examples of ACVA's findings with respect to specific buildings may help to illustrate the general points that I have attempted to make in this presentation:

1. Several years ago, four young infants died tragically at a Dutch hospital, and many more had become seriously ill. While the hospital was able to identify the cause of the infection, it could not trace the source. After an ACVA investigation, two dead bird carcasses were discovered inside the air conditioning ductwork. Bacteria from the putrified and decomposing carcasses multiplied rapidly in the dirty and damp ductwork, and had been spread throughout the building by the ventilation system.
2. After the main air handling units were replaced and high efficiency filters were installed in a large government building in Washington, ACVA was requested to confirm that the air quality was acceptable. ACVA discovered that the accumulation of dust, bacteria and fungi in the original ductwork was

re-contaminating the air supply from the new HVAC units.

3. In another instance, ACVA investigated complaints in a federal courthouse. The air handling systems were clean, and the filter systems were efficient. After extensive testing, however, ACVA discovered that textile fibers were being shed into the air from the flocked wallpaper and were causing general respiratory problems. The wallpaper was cleaned and sealed, and there were no further complaints.
4. Employees of a major bank were complaining of headaches, nausea and general respiratory problems. In that case, an electrostatic precipitator -- an ultra-efficient filter -- had been permitted to run when the air supply system was shut down. Ozone generated by the filter was diffusing up the main air supply ducts and accumulating in specific areas.
5. In 1983, physicians at the Health and Human Services Parklawn Building in Rockville, Maryland requested ACVA to investigate complaints of sick building syndrome. ACVA discovered that low efficiency and poorly installed filters had permitted unfiltered air

into office areas occupied by 5,000 - 7,000 HHS employees. In addition, recycled air rates were lower than permitted by Federal regulations, and excess fungal growth in the air handling system was discovered. Filters were upgraded, the air handling systems were cleaned and steps were taken to assure an adequate supply of fresh air. Air quality then improved substantially and complaints were virtually eliminated.

6. Students at a medical college in Maryland were experiencing respiratory problems. ACVA's investigation revealed excessive accumulations of allergenic fungi emanating from the air conditioning ductwork. The source of these fungi later was traced to sawdust produced in the mechanical room. The simple removal of the sawdust and cleaning of the mechanical room resulted in improved air quality throughout the building.

7. In another investigation, ACVA discovered excessive contamination by Cladosporium fungi within the HVAC system and ceiling voids of a 19th century four-story brownstone building being used as a law office. A total lack of fresh air, cheap and poorly installed filters

and excessive condensation from the air conditioning coils created the problem. The ventilation was increased, the system cleaned and sanitized and the filters were upgraded. There have been no more complaints of poor indoor air quality in the building. The same problem was discovered in a Pennsylvania high school that EPA had threatened to close due to high instances of staff and student sickness. ACVA took the same remedial steps described above, and the air quality within the school has been improved dramatically. EPA no longer is threatening to force the closure of the school.

8. In a state government health department, ACVA discovered that loose fiberglass insulation in the ceiling voids was being carried via the return air system to the air handling units. Poorly installed filters permitted the distribution of unfiltered air throughout the building. The problem was exacerbated by fungi in the condensate trays of the air handling units. The filters were upgraded, the air handling units were cleaned and the ductwork was sanitized. Ninety-nine percent of the complaints have disappeared, and there

has been a tremendous improvement of the building's indoor air quality.

9. ACVA investigated one hospital in which the operating room exhaust air ducts had become clogged from lint and textile fibers. Since no air could leave the room, no fresh air could enter. Pictures taken inside the exhaust ducts revealed that the ducts had bowed inward from the negative pressure created by this situation. This operating room should have received 20 air changes per hour. The ductwork was cleaned and the problem was resolved.

10. Upon investigating the ventilation system of the headquarters of a large insurance company, ACVA discovered that an exhaust duct carrying paper dust from a computer printer inadvertently had been coupled to the air supply duct for an adjacent room. In addition, excessive fungal growth was found in the paper debris in the duct. The fungus later was analyzed as Aspergillus. Over 70% of the employees in the adjacent room had become sensitized to the fungus and exhibited all the common symptoms upon exposure. ACVA disconnected the exhaust duct, removed the paper dust and disinfected

the duct work. There have been no more staff complaints.

11. ACVA recently completed one of the most interesting investigations conducted to date. A large bank requested ACVA to investigate complaints of excessive ETS in a two-year old building. The building always had been plagued with sickness problems. Another company had conducted a study through the use of subjective occupant questionnaires. Following analysis of the questionnaires, the bank was advised by that company to ban smoking throughout the building. The bank then sought a second opinion from ACVA. ACVA discovered that the ventilation system was designed poorly, and that most of the occupants would receive less than 5 cfm/person of fresh air even if the HVAC system were working to its maximum design potential. ACVA found that the fresh air dampers had been closed completely to save energy. Even if fresh air had been permitted to enter, however, the system was so inadequate that there would have been no filtration of the fresh outside air. Further, the indoor air was being recycled through grossly inefficient filters.

Substantial quantities of fungi were found in the ductwork, including Cladosporium species. The bank is now in the process of cleaning and disinfecting the ductwork, as well as increasing the ventilation and upgrading the filters.

CONCLUSION

ACVA's investigations have established that a multitude of factors contribute to poor indoor air quality. The confounding problems associated with these factors make their analysis and subsequent resolution difficult. To concentrate upon one element of this matrix, environmental tobacco smoke, would be inefficient and dangerous. It would be naive to assume that the removal or control of ETS, the most visible indoor air pollutant, would solve the indoor air pollution problems found in "sick buildings." That step ignores the many hidden sources of indoor contamination, and can lead -- at least in the short run -- to a false sense of security.

ACVA's work and experience, and evaluations conducted by NIOSH, indicate that indoor air quality can be improved significantly with proper maintenance of and attention to the HVAC system. Assuring the introduction of filtered outside air, maintaining effective filtration systems, ongoing monitoring of air quality, cleaning and repairing duct work and improving the designs of ventilation systems are necessary steps for the achievement of acceptable indoor air quality. High concentrations of environmental tobacco smoke are a symptom of the much more dangerous problem of poorly maintained and designed ventilation systems. Persistent indoor air quality complaints can be resolved only if building managers and operators are prepared to focus on those systems in an appropriate manner.

Without positive action to deal with the entire spectrum of indoor pollution, there is a grave danger that one potential irritant may take the blame for all others. In our experience, these problems are caused by a combination of factors. In practice, misguided attacks on environmental tobacco smoke actually may eliminate the one telltale marker we now have alerting us to the operation of an inadequate ventilation system. In this sense, a fitting epitaph on the tomb of a future Legionnaires' disease victim might be:

"He lived in a smoke-free environment."

Indoor air quality — the NIOSH experience

JAMES MELIUS, M.D., KENNETH WALLINGFORD, M.S., RICHARD KEENLYSIDE, M.D., and JAMES CARPENTER
National Institute for Occupational Safety and Health

Introduction

Health complaints associated with poor indoor air quality have been reported with increasing frequency among office workers over the past 10 years. A number of factors have contributed to this trend, including changes in building design to increase energy efficiency, introduction of new building materials, and increased awareness among workers of potentially toxic exposures in their work and home environments. While some exposures in the indoor environment may have serious implications for the health of the occupants (e.g., radon daughters, asbestos, formaldehyde, pathogenic microorganisms, allergens), the health risks of most indoor air exposures are poorly understood. Nevertheless, office workers who are concerned about a possible problem in their work environment often demand a thorough investigation of the environment and of its potential for health risks. For the investigator, this is commonly a challenging but frustrating experience because of the lack of guidelines and evaluation criteria for the "non-industrial" setting.

The health complaints reported by the occupants of the typical "problem building" are usually diverse and non-specific, and rarely point to an obvious cause. Also, the nature of the problem may be obscured by reports of a variety of serious, but unrelated, medical conditions associated with the office environment.

Appropriate environmental sampling is often quite difficult because of the presence of low levels of many ubiquitous substances. Specific contaminants from sources in the office can be readily measured and quantitated (e.g., ozone from copying machines), but more often very low levels of a variety of chemicals are detected. Such sampling results may in one sense be reassuring, but they have limited usefulness when linked with non-specific symptoms. These investigations are often complicated by extensive media coverage, multiple earlier investigations by other groups, and deteriorating labor relations. The "Federal expert" often comes into the picture when, despite much basic work having been done, the situation has reached a "crisis."

This paper will briefly present and review the indoor air quality investigations conducted by the National Institute for Occupational Safety and Health (NIOSH) since the start of the Health Hazard Evaluation Program. These investigations are being presented not as "THE WAY" to evaluate such problems, but, rather, to review our experience and share our insights as we have evolved our approach to these investigations.

Indoor air quality health hazard evaluations

Through December 1983, NIOSH has completed 203 Health Hazard Evaluations involving indoor air quality (IAQ) in a variety of settings (Table I). (This does not include our investigations of asbestos-related problems in office buildings.) Prior to 1978, only six IAQ evaluations were performed; however, since then, the number has increased dramatically. It appears that in the last 2 years, the number of these completed investigations has leveled off, but this change may reflect our handling of many IAQ inquiries by providing written materials and phone consultation, and by the increased capability of state and local health de-

TABLE I
Completed NIOSH Indoor Air Quality Investigations by Year (through December 1983)*

Year	Number Completed	%
Pre-1978	6	3.0
1978	9	4.4
1979	12	5.9
1980	28	13.8
1981	80	39.4
1982	44	21.7
1983	24	11.8
Total:	203	

* Does not include 81 currently active projects.

partments and other groups to handle these evaluations without our assistance.

Most of our evaluations have involved government and private offices (over 75% [see Table II]), educational institutions (14.8%), and health care facilities (9.3%). Given our mandate to evaluate occupational health problems, it is not surprising that NIOSH has not investigated very many residential IAQ problems. Thus, we do not have much experience in evaluating problems which are principally encountered in residential buildings such as exposures to radon daughters or to combustion products.

In reviewing the reports on these evaluations, we have attempted to classify our findings by the type of problem found (Table III). It is of note that nearly half of these investigations have attributed the IAQ problems to inadequate ventilation. Some form of environmental contamination was thought to be the source of the problem in approximately 30%. The source of this contamination was thought to be from inside the building in 17.7% of the investigations, outside the building in 10.3%, and from the building structure in 3.4%. Problems such as hypersensitivity pneumonitis, cigarette smoking, humidity, etc., have accounted for approximately 10% of our evaluations. Finally, in another 10%, the etiology of the IAQ problem has remained unexplained.

In reviewing these results, several factors should be considered. First, over time, NIOSH has not used a standard protocol for conducting these evaluations. Our methods and criteria have changed as we became more familiar with the problem and developed new approaches. Also, some of these investigations were conducted several years ago, leaving only scanty data and a brief report for current review. In the early studies, many of the "unknown" problems may have actually been due to inadequate ventilation, but the reports did not provide enough information to determine this. Thus, there may be considerable misclassification in this list.

TABLE II
Completed NIOSH Indoor Quality Investigations by Building Type (through December 1983)

Type	Number	%
Government and business offices	154	75.9
Schools and colleges	30	14.8
Health care facilities	19	9.3
Total:	203	

TABLE III
Completed NIOSH Indoor Air Quality Investigations by Type of Problem (through December 1983)

Problem	Number	Total
Contamination (inside)	36	17.7
Contamination (outside)	21	10.3
Contamination (building fabric)	7	3.4
Inadequate ventilation	98	48.3
Hypersensitivity pneumonitis	6	3.0
Cigarette smoking	4	2.0
Humidity	9	4.4
Noise/illumination	2	1.0
Scabies	1	0.5
Unknown	19	9.4
Total:	203	

This listing is also not necessarily representative of the general distribution of indoor air quality problems in offices. Often, NIOSH is requested to conduct an evaluation only after initial attempts to identify the problem have failed, or complaints have persisted after initial corrections have been made. Large public-sector agencies use NIOSH as a resource, but managers of smaller offices or office buildings may be unaware of our program. Therefore, these facilities may be underrepresented on our list. Despite these shortcomings, the list does provide an overview of the types of indoor air quality problems encountered in office environments. A brief review of the major types of IAQ problems follows.

Contamination from inside the office environment

This classification (approximately 18%) refers to exposure to a chemical or other toxic agent generated within the office space. Usually the symptoms experienced by the office worker are directly linked to the exposure, but, if the exposure is disseminated through the building's ventilation system, localization of the source may be difficult. Examples from Health Hazard Evaluations include exposures to methyl alcohol from spirit duplicators,[1] exposures to methacrylate from copiers,[2] and exposures to sulfur dioxide from a heating system.(3) Occasionally, a very specific health effect can aid in the investigation, such as the occurrence of dermatitis from amines used in humidification systems.(4,5) The use of pest control agents, such as chlordane, may also cause persistent problems in office environments.(6,7)

These problems can usually be identified by inspecting the affected office area (e.g., copying machines) and by questioning about other uses of chemicals in the building (e.g., pesticides, humidification agents). Environmental and medical testing appropriate for that chemical can then be conducted.

Contamination from outside the building

This classification (approximately 10%) refers to exposure to a chemical or other toxic substance originating from a source outside the building. Common examples include motor vehicle exhaust either from a parking garage or loading dock entering the building through the intake from the ventilation system.(3,8) Many of these exposures are "presumed" explanations that cannot be documented at the time of the investigation. Although the dispersal of substances through the ventilation system may seem obvious, the complexity of the system may require the use of more sophisticated techniques such as a tracer gas to document the problem.(9) Other outside sources include nearby construction activity which may generate sufficient exhaust fumes, dust, or other contaminants to cause complaints in nearby offices.(10)

A few years ago, NIOSH evaluated a dramatic case of outside contamination of an office building when we discovered a 6-million-gallon underground gasoline spill while investigating irritative symptoms among the office occupants.(11) While this may be viewed as unusual, there is concern about the increasing number of underground gasoline tank leaks.

Contamination from the building fabric

Contamination from the building fabric (approximately 3% of our evaluations) refers to problems from the material used to construct the building. While not included in our list of evaluations, asbestos is obviously a major source of concern about indoor air quality. Other insulating materials are a common source of these problems because of the release of substances such as formaldehyde.(12) Dermatitis due to fibrous glass has also been a common problem, usually after the fibrous glass insulation has been disturbed during some construction activity.(13)

Hypersensitivity pneumonitis

This group (approximately 3% of our evaluations) refers to problems due to a hypersensitivity reaction to microorganisms in the building environment. Although not a common cause of office problems, the potential medical severity of this condition and the difficulties in controlling this problem make it an important cause of office problems. NIOSH's evaluations regarding this problem will be discussed later in this volume.

Inadequate ventilation

By far the largest classification, this group makes up approximately one-half of our completed evaluations. Our determination of inadequate ventilation is commonly made after considering a number of factors, including the absence of other sources of contamination and the presence of only very low levels of contaminants in our environmental sampling results. These, linked to the widespread occurrence of non-specific symptoms such as headaches, eye irritation, and upper respiratory irritation, suggest that an evaluation of the ventilation system may be warranted.

The evaluation of ventilation systems will be discussed later. Our methods range from obtaining specifications on the building ventilation system to detailed air flow measurements. Both approaches can present difficulties. Information on the specifications of the ventilation system is often not readily available from the building operators. Buildings with renovated ventilation system are often very difficult to evaluate because of the piecemeal approach used in such renovation. The ASHRAE guidelines for ventilation are usually used for comparison.(14) While one can question the basis for these guidelines, we have found them useful in evaluating IAQ problems and for recommending corrections where there is a problem because of inadequate ventilation.

The pathogenesis of complaints or symptoms caused by inadequate ventilation is not clear, but certain extreme situations have provided us with some insight into the relationship between such complaints and inadequate ventilation. In 1982, we conducted an evaluation at a government office building in Idaho with widespread complaints among the employees. Despite environmental surveys showing no significant contaminant levels, the employees were moved to another building. Our investigators found that the air intake for the building had been covered with plastic one year earlier, to protect the air handling system from airborne debris from Mount St. Helens, and that this cover had never been removed. Removal restored intake of adequate outdoor air to the building and allowed reoccupancy without significant problems.(16) However, in this situation, no environmental measurement indicated that there was a problem. In general, we have not found any environmental measurement to be useful as an indicator of poor ventilation. However, it should be noted that other investigators have found carbon dioxide levels useful for such evaluations.(16,17) Low levels of multiple contaminants are often present in these situations and are currently the best explanation for the occurrence of symptoms. The pattern of contaminants probably varies from building to building, but we do not yet have adequate measurement techniques or adequate knowledge to easily recognize this problem through environmental sampling.

Current evaluation methods

Our current approach to evaluating office environment requests usually begins with a walk-through evaluation by an industrial hygienist. Prior to this, we try to obtain background information on the history of the building design or construction and try to ensure that the building engineer will be present during the investigation. During the initial visit, we obtain a history of complaints among office occupants by interviewing as many as is feasible. This is helpful not only for identifying the type of medical complaints, but also for obtaining a chronology of the problem and ascertaining the time pattern of symptoms (afternoon more than morning, etc.).

Potential sources of contamination are identified during the initial walk-through evaluation. Some will be obvious (e.g., copying machines), while others may be identified only after careful questioning (e.g., pesticide spraying). We also usually inspect the ventilation system for the particular office area and attempt to understand its connection to the system for the entire building. Information on the control of the ventilation system (outdoor air intake relative to temperature, etc.) is also obtained. Some environmental sampling may be conducted if a source of contamination is found or suspected. Some general air monitoring (e.g., organics) may also be conducted, but this is usually more helpful to reassure the occupants that the toxic substances of concern to them are not present in any degree than it is for identification of a problem.

The problem may be resolved during this initial visit, but, in some instances, more extensive environmental sampling, a medical study, a ventilation assessment, or some combination of these may be required. These may be necessary either to better identify the source and extent of the problem or to alleviate the concerns of the affected employees. Once our investigations are complete, our findings and recommendations are communicated to the involved parties.

Future activities in indoor air quality

More research into office ventilation and its effect on background levels of contaminants is necessary to provide better guidelines for evaluating and controlling indoor air quality problems. Because of the nature of these exposures, a variety of governmental agencies and private groups are involved in this research effort. Recently, the Environmental Protection Agency, the Consumer Product Safety Commission, the Department of Energy, the Department of Health and Human Services, the Tennessee Valley Authority, and several other Federal agencies formed a coordinating committee on indoor air quality research. This committee will help to coordinate Federal government indoor air quality research. Already, an inventory of IAQ-related research in the Federal government has been prepared. These agencies are also coordinating the planning of a possible large national survey of indoor air pollution and related health problems.

While much of this effort and related research may seem remote to investigating specific problems in office buildings, they may provide the basis for better guidelines for evaluating indoor air quality and for necessary corrective steps. At the same time, improved methods of assessment are needed to evaluate specific types of problems.

NIOSH is currently developing better methods for assessing indoor air quality (e.g., microorganism levels, ventilation parameters, etc.). Other groups are working on methods for other types of assessments. Meanwhile, we will continue to evaluate indoor air quality problems and, hopefully, continue to improve our efforts in these assessments.

References

1. Centers for Disease Control: Methyl Alcohol Toxicity in Teacher Aides Using Spirit Duplicators — Washington. *Morbidity & Mortality Weekly Report* 29:437-438 (1980).
2. National Institute for Occupational Safety and Health: *Congressman Cavanaugh's Office.* Health Hazard Evaluation Report No. HETA 80-067-734. NIOSH, Cincinnati, OH (1980).
3. National Institute for Occupational Safety and Health: *Wappingers Falls School.* Health Hazard Evaluation Report No. HETA 83-172-1409. NIOSH, Cincinnati, OH (1984).
4. National Institute for Occupational Safety and Health: *Boehringer-Ingelheim, Ltd.* Health Hazard Evaluation Report No. HETA 81-247-958. NIOSH, Cincinnati, OH (1981).
5. National Institute for Occupational Safety and Health: *Cornell University.* Health Hazard Evaluation Report No. HETA 83-030-1351. NIOSH, Cincinnati, OH (1983).
6. National Institute for Occupational Safety and Health: *Georgetown University.* Health Hazard Evaluation Report No. HETA 83-444. NIOSH, Cincinnati, OH (under study).
7. National Institute for Occupational Safety and Health: *Planned Parenthood.* Health Hazard Evaluation Report No. HETA 83-258. NIOSH, Cincinnati, OH (under study).
8. National Institute for Occupational Safety and Health: *Department of Justice.* Health Hazard Evaluation Report No. HETA 80-024-887. NIOSH, Cincinnati, OH (1981).
9. National Institute for Occupational Safety and Health: *Cincinnati Technical College.* Health Hazard Evaluation Report No. HETA 82-288-1341. NIOSH, Cincinnati, OH (1983).
10. National Institute for Occupational Safety and Health: *McCalls Publishing Company.* Health Hazard Evaluation Report No. HETA 81-087-1021. NIOSH, Cincinnati, OH (1981).
11. Centers for Disease Control: Employee Illness from Underground Gas and Oil Contamination — Idaho. *Morbidity & Mortality Weekly Report* 31:451-453 (1982).
12. National Institute for Occupational Safety and Health: *Tri-Valley Federal Credit Union.* Health Hazard Evaluation Report No. HETA 81-108-883. NIOSH, Cincinnati, OH (1981).
13. National Institute for Occupational Safety and Health: *Ellis Hospital.* Health Hazard Evaluation Report No. TA 80-090. NIOSH, Cincinnati, OH (1980).
14. American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.: *ASHRAE Standard 62-1981. Ventilation for Acceptable Indoor Air Quality.* ASHRAE, Atlanta, GA (1981).
15. National Institute for Occupational Safety and Health: *U.S. Forest Service.* Health Hazard Evaluation Report No. HETA 81-150-964. NIOSH, Cincinnati, OH (1981).
16. Rajhans, G.S.: Indoor Air Quality and CO_2 Levels. *Occupational Health in Ontario,* pp. 160-167. P.L. Pelmear, Ed. Ontario Ministry of Labour (1983).
17. Hicks, J.B.: Tight Building Syndrome: When Work Makes You Sick. *Occup. Safety & Health,* pp. 51-57 (1984).

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF
EXTERNAL AFFAIRS

Honorable James H. Scheuer
Chairman
Subcommittee on Natural Resources,
Agriculture Research, and Environment
Committee on Science and Technology
House of Representatives
Washington, D.C. 20515

Dear Mr. Chairman:

Enclosed are responses to questions you sent to us on November 3, 1986 following the September 17, 1986 hearing on indoor air quality and radon. I apologize for our late response.

I am not able to include at this time our response to question 3 of the set of questions on indoor air quality, nor our responses to questions 2,6,9, and 13 of the set of questions on radon. I will forward those responses within the week.

I thank you for your patience in this matter. If I can be of further assistance, please let me know.

Sincerely,

Joy Wilson

Jennifer Joy Wilson
Assistant Administrator
for External Affairs

INDOOR AIR QUALITY

Question 1: The 1986 EPA Appropriations Committee Report directed that the annual program plan and project funding schedule of each agency belonging to the Interagency Committee on Indoor Air Quality (CIAQ) be reviewed and approved by the CIAQ. Does EPA submit its annual indoor air research budget to the CIAQ for comment and approval? If so, when in the budget cycle does this occur? For FY-86, the funding was not available until April 1986.

Answer: EPA's indoor air research program plan is based on direction for research from Congress. EPA drafts a research program plan to address these requirements and obtains comments from CIAQ members and CIAQ workgroups. This provides an opportunity to review Congressional direction for indoor air research, avoid duplication of research efforts in other Federal agencies and collaborate on areas of mutual interest. Comments are solicited during the first quarter of the fiscal year or when funds become available.

Question 2: According to the CIAQ "Research Plan", EPA also conducts health effects research out of its "base" program relevant to indoor air issues. Does EPA submit the portion of its base program which is relevant to indoor air issues to the CIAQ for its comment and approval?

Answer: EPA does not submit its relevant base program plan for health research to the CIAQ for review and comment. EPA does, however, inform CIAQ members about the status and progress of its base program research in health and in other research areas. The Agency's indoor air effort augments the base program research, thereby making more efficient use of indoor air research resources provided by Congress.

Question 4: Did each of the co-chair agencies have an opportunity to review the final draft of the response to Chairman Boland before it was sent to him?

Answer: Each of the co-chair agencies had an opportunity to review the draft response before it was sent to Congressman Boland. In fact, they and their staffs were very much involved in working on preparing the response.

Question 5: Are there any projects which have been jointly planned or funded through the Committee on Indoor Air Quality?

Answer: Several projects have been jointly planned or funded by the CIAQ. For example, specific indoor air research experiments in the Harvard Six Cities Study were co-funded by the member agencies, and in efforts to develop data bases on sources of indoor air pollutants, CIAQ members have collaborated in several key areas. CIAQ members interested in indoor air models have pooled their resources to fund research conducted by the National Bureau of Standards. CIAQ members also worked with EPA to prepare an indoor air questionnaire for inclusion in the NHANES III survey. They are jointly planning a workshop, sponsored by the Interagency Task Force on Environmental Cancer and Heart and Lung Disease, on the health effects of indoor air pollution, to be held in 1987.

Question 6: EPA has previously said that it intends to work with HHS to obtain information on indoor air quality and health effects through the National Health and Nutrition Evaluation Survey (N-HANES) scheduled to begin in FY 87. EPA apparently deleted a discussion of the N-HANES field study from its description of Task 6 before sending the report to Congress. Why was this discussion deleted? What is the status of the N-HANES activity?

Answer: On August 11, the Administrator of EPA affirmed that the Agency should participate in the next National Health and Nutrition Examination Survey (NHANES-III). Prior to that time (while the Boland response was being prepared), EPA's proposed program to participate in NHANES-III was in the formative stages. It would have been premature to indicate the extent to which the Agency was prepared to participate in the survey at the time the report was sent to Congress.

Subsequent to the Administrator's decision, a Stage I Proposal to participate in NHANES-III has been sent by EPA to the National Center for Health Statistics (NCHS). In accordance with NCHS guidelines, the Stage I proposal merely contains statements of the general aims and public health significance of the proposed areas of research recommended by EPA for inclusion in NHANES-III. A Stage II proposal containing detailed information on test protocols, quality assurance plans, interagency consortia, analysis plans and budgets is currently being prepared for those areas that NCHS believes to be feasible for inclusion into the survey.

One area proposed by EPA--acting in conjunction with several other agencies on the Committee on Indoor Air Quality (CIAQ)--recommends that NHANES-III obtain information through passive monitoring and questionnaires on factors that affect indoor exposures to various pollutants including tobacco smoke. This information, along with pulmonary function tests and reported histories of respiratory illnesses that would be obtained through NHANES-III, would be used to ascertain relationships between respiratory health status, the quality of indoor home environments, smoking behavior, and other relevant variables that affect respiratory health. A Stage II proposal for this area is currently being prepared and negotiated with the National Center for Health Statistics.

QUESTION 7: EPA sets out its "strategic approach" to indoor air pollution on pages 29-32 of the Research Plan. While it details research methodology, nowhere does it address the purpose for which the research is being conducted. What are the policy decisions to be made that will use this data, and how will the data be relevant to those decisions?

ANSWER: The Office of Air and Radiation is developing Agency policy and strategy on indoor air quality. Several key decisions have been made. OAR will develop an indoor air program based on disseminating information and guidance, as authorized under Title IV of the Superfund Amendments and Reauthorization Act of 1986; however, other program offices may use authorities which they administer to take regulatory or non-regulatory actions to reduce exposure to indoor air pollution if they determine that risks warrant action.

The goals of the indoor air program are to define the nature and magnitude of problems posed by indoor air pollution and to reduce the risk to human health and productivity from exposure to indoor air pollution. In pursuing these goals, EPA will look both at individual pollutants and products which cause exposure and at building environments where exposure occurs.

Research conducted by ORD will help give EPA the scientific basis on which to make policy decisions on: 1) which pollutants, products, and building environments should be included in the OAR indoor air information dissemination program, 2) which pollutants and products should be referred to other program offices or other Federal agencies for consideration for actions, and 3) how to assist the public and private sector in diagnosing, assessing, and mitigating or preventing indoor air quality problems.

Question 8: According to the Research Plan, EPA planned to spend $700,000 on field survey work in FY 1986. How much did EPA spend on such surveys in FY 1986, and when were the contracts for such work awarded?

Answer: In FY 86, EPA spent $495,000 for design and methodology projects that support indoor air field studies. This is described further below in question #12. No money was actually expended on surveys. As a result of a recent SAB review of ORD's indoor air research program, remaining funds are currently being redirected from "surveys" and will be used to improve diagnostic procedures for assessing indoor environments.

Question 9: In his testimony, Mr. Ehreth stated that EPA is presently reevaluating the indoor air quality program. What is the schedule for completing this evaluation? Does EPA plan to have federal, state and public involvement in this process? What form will EPA's conclusions of its evaluation take? Will it include a report to Congress?

Answer: The schedule for re-evaluating the EPA indoor air quality research program in a Research Needs Assessment document is as follows:

October 3, 1986	Organizational Meeting: Formation of Steering Committee
October 6, 1986	Preliminary Outline for Research Needs Document Formulated
November 10, 1986	Plan workshop for review of RNA document with Harvard School of Public Health
December 18, 1986	Preliminary in-house review of RNA in advance of workshop.
January 19-23, 1987	Workshop to review RNA document: Harvard University
March, 1987	Revised RNA document complete
March, 1987	Long-term Research Plan based on research needs assessment, integrated with Agency policy.

Federal agencies, various public interest groups, and university researchers are involved in the preparation and review of the research needs assessment document. This document will be in the form of two volumes, the first of which will be an assessment and summary of information known about indoor air, as well as an identification of knowledge gaps; the second will be a research strategy which will be integrated with the first volume. A report to Congress on the research program will be provided, as required by amendments to Superfund passed by Congress in October 1986.

Question 10: Please provide a breakdown of how the $2.2 million Congressional add-on for FY 86 indoor air research was spent.

Answer: The following table shows the distribution of funds for the $2.2 million FY-86 Congressional add-on for indoor air research.

Engineering Projects

Funds

R&D	S&E	FTE	
$220,000	72,000	1.4	Chamber studies of Organic Emissions from Material Sources
50,000	42,000	.8	Chamber studies of Organic Emissions from Unvented Combustion Sources
20,000	82,000	1.6	Indoor Source Emissions Data Base
50,000	42,000	.8	Engineering Evaluations of Air Cleaners for Indoor Particles
Health Projects			
$130,000			Bioassay Studies of Indoor Organic Pollutants: Combustion Emissions and Building Materials
20,000			Biochemical/Biological Markers for Human Exposure Assessment and Dosimetry of Environmental Tobacco Smoke and Other Indoor Combustion Emissions
100,000			Neurobehavioral Effects of Indoor Air Pollutants
Monitoring Projects			
$214,000			Initiation of Proposed Indoor Air Quality Field Study and Related Methods Development
184,000			Methods Development for Indoor Air Quality Field Study
97,000			Preparation of Survey Questionnaires and Performance of Survey Methodology Refinement Studies
55,000			Support for Technology Transfer (1986 Consumer Federation of America IAQ Conference; 1987 International IAQ Symposium, Berlin)
87,000			ORNL Microenvironment Study
15,000			Nicotine Study Included with NYERDA IAQ Study
2,000			Nicotine Analyses by University of New Mexico
100,000			IAQ Model Development through NBS-IAG
60,000			Support for CIAQ
5,000			Support for Indoor Air Research Needs Document
25,000			Workshop Review of Indoor Air Research Needs Document
566,000			Support for Additional Indoor Air Research Projects to be planned and funded in FY-87.
$2,000,000	238,000	4.6	TOTALS
2,238,000			Total Expenditures of FY-86 Funding

Question 11: How much did EPA pay a contractor to prepare the Research Plan?

Answer: EPA paid SRA Technologies of Alexandria, VA $52,000 to support the CIAQ and its planning activities during the last six months of FY 1986. Of that amount, preparation of the Research Plan transmitted to Congressman Boland cost EPA $23,000. The CIAQ is reviewing options to update the plan for status reporting.

Question 12: What is the status of EPA's plans to conduct a national multi-pollutant field survey, or a pilot survey? Is any work proceeding on the survey or pilot survey pending completion of the SAB review? How much has EPA already spent or committed on such a survey?

Answer: In accordance with Congressional direction that we conduct a national survey, ORD spent $495,000 to design the survey and to develop monitoring methods for use in such a survey. As a result of a recent Science Advisory Board review of ORD's indoor air research program, we did not conduct a national multi-pollutant field survey. Instead, the research will be redirected to develop and field test the diagnostic procedures for monitoring sources of indoor air pollution and assessing their health effects.

QUESTION 13: Please describe the role that the Office of Air and Radiation will play with respect to indoor air quality issues, and the relationship between OAR and ORD's indoor air quality activities.

ANSWER: As stated above, the Office of Air and Radiation is charged with developing indoor air policy and a non-regulatory program to implement that policy. OAR will carry out screening and assessment activities so that Agency actions are targeted to pollutants, products, and building environments that are a significant risk to human health or productivity. OAR will also develop an information dissemination program to assist the public and private sectors to protect and maintain indoor air quality.

There are several ways that the indoor air quality activities of OAR and ORD relate to each other. OAR is preparing guidance for ORD as part of the development of the indoor air quality policy and strategy. In the portion of the Research Needs Assessment (RNA) that lays out the multi-year planning effort, ORD will show how the RNA meets the policy goals and program needs as developed by OAR. OAR and ORD will coordinate joint planning for the research program through the indoor air subcommittee of the Air and Radiation Research Committee. ORD data and findings will be a primary source of information as OAR undertakes its information dissemination program.

QUESTION 14: What is EPA's plan for disseminating indoor air quality information to the States?

ANSWER: EPA believes that States and local governments are on the front line in addressing public concerns about indoor air quality. Therefore a primary objective of the EPA indoor air effort will be to enhance the capability of State and local governments to: 1) deliver information and technical assistance; 2) perform building investigations; 3) oversee products and services provided by the private sector; and 4) enact and enforce laws that affect the way buildings are designed, constructed, and operated.

EPA will develop an information dissemination program that helps States and local govenments build the skills and technical expertise necessary to perform these tasks. EPA will sponsor several meetings with State and local officials to find out how they characterize their needs and concerns. EPA anticipates that States and local governments will ask that the EPA programs include information, protocols and guidance, training, and technical assistance. EPA will provide Congress a description of the information dissemination program as part of Part II of the Report to Congress, required by Title IV of the Superfund Amendments and Reauthorization Act of 1986. EPA plans to submit Part II next April.

Question 15: Other than radon, what indoor air pollutants present the greatest threat to public health?

Answer: It is not scientifically possible to assign a "greatest threat" ranking to any list of pollutants since the conditions of exposure for each pollutant can vary from insignificant to highly detrimental depending on many variables found in indoor spaces. Chemicals on the following list, depending on conditions and circumstances, have been identified with health problems in indoor air. The problems can range from lethality to chronic damage to organs and organ systems, as well as to reversible health changes which can interfere with well being and productivity. Further, there are major uncertainties in knowledge of the exposure-health response relationship, especially as it concerns chemical mixtures.

With these considerations stated, the following are indoor pollutants, other than radon, which present a potential threat to public health:

- products of incomplete combustion, including:
    - particulates
    - carbon monoxide
    - organics which include polycyclic aromatic hydrocarbons, semi-volatile and volatile, polar and non-polar compounds
    - formaldehyde
    - environmental tobacco smoke
    - woodstove emissions
    - kerosene heater emissions
- nitrogen dioxide
- volatile organic compounds (VOC)
- fibers, including asbestos
- biological contaminants
- pesticides

RADON QUESTIONS

1. Please provide an explanation of the basis for the risk estimates included in the chart contained in "A Citizen's Guide to Radon," and specify any assumptions that were made in order to extrapolate the data from the uranium miners' studies to the adoption of the .02 Working Levels "action standard" for residential radon.

The bases for the risk estimates included in the chart contained in "A Citizens Guide to Radon" are as follows:

Radon: Based on a review of thirteen epidemiological studies of miners exposed to radon, current scientific consensus on the use of absolute versus relative risk projection models, and the advice of the EPA Science Advisory Board, relative risk factors of 1% increase in lung cancer per WLM exposure and 4% increase in lung cancer per WLM exposure were selected as appropriate limits to the range of risk coefficients for the US population.

These relative risk factors were used with 1980 age-specific mortality rates and the 1980 lifetable in an actuarial type relative risk projection model. The excess lung cancer mortality expected in a cohort of 100,000 persons born in 1980 and exposed for life was calculated for the various exposure levels listed in the chart.

Chest X-rays: An actuarial type relative risk projection of estimated lung cancer was made based on an FDA estimate of radiation dose per chest x-ray, the relative risk of lung cancer per rad taken from BEIR-3, 1980 age-specific mortality rates and the 1980 lifetable in a calculation analogous to that made for the radon estimate.

Smoking: The distribution of lung cancer risks attributable to smoking was taken from the Surgeon General's report on "Smoking and Health" for 1975 and "The Health Consequences of Smoking" for 1980 and 1982.

The assumptions made in extrapolating from miner data to a general population are:

a. The increased risk of lung cancer in miners is associated solely with radon daughter exposure; no other factors in the mine have a significant impact on the risk of lung cancer.

b. The results observed in miners are representative of what would be expected in adult males at similar levels of exposure.

c. Risk expected per unit exposure in males and females is similar.

d. Risk expected per unit exposure in children is similar to that in adults.

e. Risk is proportional to amount of radon daughters deposited in the airways and therefore to the amount inhaled and the lung size.

f. The interaction of radon daughter exposure and cigarette smoking is the same in miners and in members of the general population.

g. The risk factors are assumed to be valid at the generally lower exposure rates observed in housing .

Question 3: Did other Federal agencies review EPA's proposed risk estimates prior to publication? Were any concerns or disagreements expressed about the proposed risk estimates? What were those concerns?

A draft of "A Citizen's Guide to Radon" was sent for review to the Radon Workgroup of the Committee on Indoor Air Quality and to member agencies of the Radon Subpanel of the Committee on Interagency Radiation Research and Policy Coordination. Comments were received from approximately 13 Federal agencies, including the Office of Management and Budget, the Centers for Disease Control, and the Department of Energy. The draft of "A Citizen's Guide" was also reviewed by the lead health, environmental, and radiation protection officials in each State.

Commenters raised several questions about the risk estimates presented in "A Citizen's Guide." The Department of Energy and the Office of Science and Technology Policy (OSTP) raised the concern that the range of EPA's risk estimates for radon exposure was too high and that they included values higher than the estimates of the National Council on Radiation Protection and Measurements (NCRP). Comments from these two agencies went on to suggest that EPA reduce its risk estimates and admit greater uncertainty in the estimates of risk at lower exposure levels. Other Federal agencies did not specifically comment on the validity of EPA's risk estimates; instead, they focused on the presentation of those risk estimates in the brochure.

EPA took into consideration the comments made by the Department of Energy and the Office of Science and Technology Policy. Rather than abandon its range of risk estimates, which is based on the recommendations of EPA's Science Advisory Board, the Agency chose to clarify the origin of its estimates and include a discussion of the uncertainties associated with all estimates of risk from radon exposure. We note that the report entitled, "Radon Protection and Health Effects," issued in August 1986 by OSTP's Radon Subpanel listed the same range of observed risk values as chosen by the EPA Science Advisory Board. The consensus report was developed by a team of scientists from numerous Federal agencies.

Question 4: The National Council on Radiation Protection recommends a "remedial action" level at 8 picocuries per liter, twice the EPA level. The Bonneville Power Authority recommends remedial action at 5 picocuries per liter. Why is there a difference in these recommendations?

In order to understand the reasons for the differences in the "remedial action" levels proposed by the National Council on Radiation Protection and Measurements (NCRP), the Bonneville Power Administration (BPA), and the Environmental Protection Agency (EPA), it is important to examine the context in which each was established. In selecting its remedial action level of 2 working level months (WLM) (approximately 8 pCi/l), the NCRP acknowledged that "the remedial action level selected must be based on consideration of the probable distribution of natural exposures and of the calculated risk associated with exposures at that level. In particular, recommendation of a low level to minimize the risk is not warranted if the impacts are excessive." (NCRP Report No. 77, "Exposures From the Uranium Series with Emphasis on Radon and Its Daughters, March 15, 1984, p. 88.) The NCRP went on to say, "...it is considered that an excess risk of death from lung cancer of 2% or more over a lifetime for the individual exposed to elevated or enhanced levels of radon daughters should be avoided." (Ibid.) In other words, in selecting a remedial action level of 2 WLM, NCRP weighed the associated risk against the probable number of homes affected, and decided it was willing to accept a 2% lifetime risk to minimize the number of homes that could potentially require remedial action.

The Bonneville Power Administration in selecting its remedial action level of 5 pCi/l made a similar type of determination. In its Record of Decision on Its Expanded Weatherization Program, BPA explains that its selection of 5 pCi/l was based on "...(1) a review of proposed standards, guidelines, or recommendations suggested by various agencies or organizations for controlling health hazards associated with airborne radon in residences; (2) an analysis of the regional health effects from radon, the uncertainty surrounding the level of health effects, and the relative accuracy of the proposed monitoring and mitigation strategy; and (3) a review of program economics, including cost of mitigation and estimates of the number of residences which exceed the action level." (Department of Energy, Bonneville Power Administration, Record of Decision, The Expanded Residential Weatherization Program, October 1984, p. 19.) Again, risk was weighed against the potential number of houses affected.

EPA's recommended level of 4 pCi/l was established as the lower end of a four-tier scheme which provides homeowners with recommendations as to whether and how quickly they should take remedial action based on the radon concentration in their homes.

In this context, 4 pCi/l is seen as a target for remedial action. In other words, levels above 4 pCi/l do not necessarily demand remedial action, but because of the availability of reduction methods, consideration should be given to reducing such levels.

In selecting 4 pCi/l as a remedial action target, EPA weighed the associated risks against current technological capability. It appears that 4 pCi/l represents the lower bound of results achievable by today's technology. Based on the range of the risk estimates provided by EPA's Science Advisory Board, this value represents approximately a 2% lifetime risk of lung cancer.

The differences in the remedial action levels proposed by NCRP, BPA, and EPA can be attributed primarily to differences in the context in which each level was established.

QUESTION 5: What "remedial action" limits have other countries established?

A number of European countries have conducted national radon surveys, including the United Kingdom, Ireland, Germany, France, Italy, Denmark, Belgium and Sweden. A few of these countries have established guidelines for radon exposures in residential structures. In addition, the International Commission on Radiation Protection (ICRP) has also proposed guidelines for indoor radon exposures. Summarized below are the standards or guidelines established by the ICRP, Canada, Sweden, and the United Kingdom. These countires generally express their guides in different units so they have been converted to their approximate concentration in picocuries per liter of air (pCi/l).

ICRP

Remedial Action in Existing Houses

Relatively Easy	10 pCi/l
Severe and Disrupting	Larger Volue Might be Sufficient

New Houses

Upper Bound	5 pCi/l

CANADA

Remedial Action in Existing Houses	20 pCi/l
Design Limit for New Houses	None

SWEDEN

Remedial Action in Existing Houses	20 pCi/l
Remedial Action in Renovated Houses	10 pCi/l
Limit for New Houses	4 pCi/l
Design Objective for New Houses	About 1 pCi/l

UNITED KINGDOM - Proposed by the National Radiation Protection Board

Remedial Action in Existing Houses	20 pCi/l
Design Limit for New Houses	4 pCi/l

QUESTION 7: What is the basis for the EPA estimate that 12% of dwelling units are above .02 working levels? Does that estimate include apartments as well as single-family homes?

EPA's estimate that about 12% of homes across the country have indoor radon decay product concentrations above 0.02 working levels is primarily based upon analyses of the data summarized in a 1984 report from the Lawrence Berkeley Laboratory (LBL) titled "Distribution of Airborne ^{222}Radon Concentrations in U.S. Homes." Although the number of homes considered in this study was small (552 are included in the summary distribution), its authors included data from several surveys across the country. This study has since been expanded by LBL to include approximately 1400 homes. The distribution of levels in the original study have been supported by this expansion.

EPA's estimate of 12% of homes exceeding 0.02 working levels is based on the raw data included in the LBL study. Some scientists, who have also analyzed this data, have assigned weighting factors to various components of this data. Following this approach, one scientist estimated that approximately 7% of U.S. homes would exceed 0.02 working levels. Other estimates of the number of homes that might exceed 4 pCi/l range from approximately 5% to approximately 15%. It is this uncertainty that highlights the need for a national assessment of radon levels aimed at accurately determining the distribution of levels across the country.

EPA's estimate applies to only single-family homes, since that is the focus of the LBL study. Apartments, particularly high-rises, should exhibit lower average concentrations than single-family homes or other dwellings that have direct contact with the ground.

Question 8: What percentage of homes are likely to have levels of radon above 20 picocuries per liter? Are such homes likely to be distributed purely randomly?

Answer: Since we do not yet have the results of a national survey, it is not possible to state the percentage of homes with radon levels in excess of 20 pCi/l. Extrapolating from the data suggests that the number of houses probably ranges from about 100,000 to over 1 million.

However, it is certain that such homes will not be distributed randomly. Instead, they will be concentrated in areas in which geologic conditions are particularly likely to cause substantial amounts of radon to be present in the rock soil surrounding homes. Such conditions are present in the Reading Prong formation in Pennsylvania, for example, where more than 10 percent of the 17,500 homes that the State has surveyed have radon concentrations above the 20 picocuries per liter in the lowest liveable level of the house.

RADON PROGRAM

Question 10: Please describe the status of EPA's radon mitigation demonstration program, including results achieved in New Jersey and Pennsylvania.

Answer: In August 1986 EPA published Radon Reduction Methods - A Homeowner's Guide and Radon Reduction Techniques for Detached Houses - Technical Guidance, based on its experience with the 18-home project in Pennsylvania and a review of other available information. These publications will be updated in 1987.

As of October 1986, EPA tested 46 installations in 30 houses in eastern Pennsylvania. Initial radon levels in these houses ranged from 20 pCi/l to nearly 1500 pCi/l. Currently, 14 houses have been reduced to below 4 pCi/l, 24 houses (cumulatively) are less than 20 pCi/l and 28 of the 30 houses are below 40 pCi/l. (Installations in the remaining two houses have not yet been completed and tested.) In percent reduction terms, performance of the installations in the 30 houses is as follows: 90% in 13 houses; 75% in 22 houses; 50% in 25 of the houses. Concentrations in three of the houses have been reduced less than 50% and have not yet been measured in two of the houses. In Phase III of this project, which is now underway, 24 installations will be made in some of these houses to further reduce the radon levels and in 10 additional houses.

In Clinton, New Jersey, the mitigation of 10 houses with very high initial radon levels is largely completed. All ten of the houses had initial radon levels exceeding 400 pCi/l and four of the 10 houses had initial levels that exceeded 1000 pCi/l. As of October 1986, all of the houses were reduced to less than 20 pCi/l and three houses (including the two with the highest levels) were reduced to less than 4 pCi/l. Percentage reductions in all ten houses have exceeded 95%. The real test of these reductions will occur during the coming winter months, when the driving forces for indoor radon contamination are usually at their highest. EPA will be monitoring the radon levels in these houses during this period.

At the State's request, EPA also prepared evaluations of 20 additional houses in Clinton to assist other area homeowners in installing appropriate mitigation measures. Each evaluation was given to the homeowner and all 30 evaluations (with the names removed) were made available to any interested Clinton homeowner. During the course of this project, EPA, in conjunction with the New Jersey Department of Environmental Protection and the mayor of Clinton, held 5 town meetings to discuss the research project and its findings with the community.

EPA currently has two other projects underway. One is in conjunction with the New York State Energy Research and Development Authority (NYSERDA) with cooperation by the NYS Department of Health and the NYS Energy Office. Eight houses have been selected in Putnam and Orange counties for mitigation this fall; a similar number will be selected in the Albany area in January 1987. EPA is also conducting a study in the piedmont area of New Jersey in conjunction with the New Jersey Department of Environmental Protection and co-funded by the U.S. Department of Energy to develop diagnostic protocols for studying radon entry into structures.

By the end of calendar year 1986, EPA will have conducted radon mitigation research in approximately 55 existing homes. During calendar year 1987, EPA plans to conduct research in approximately 110 additional existing houses and 25 new houses. The demonstrations will continue in Pennsylvania, New Jersey and New York and will begin in other States.

Question 11: How does the Agency assist States in developing efforts to conduct radon surveys? How many States has the Agency assisted and what are the Agency's plans for assisting other States which have asked for assistance? What role do the State survey efforts play in EPA's planned national survey?

Answer: In response to requests from States, the Agency has initiated a program to provide several different types of assistance to States that want to conduct indoor radon surveys. This can include radon detectors and the associated laboratory analyses as well as assistance with survey design and sampling procedures, geological characterization of different areas of the State, and data management and evaluation. EPA has announced that we will help 10 States conduct surveys this winter, when screening measurements for elevated radon levels are most reliable. Another 11 States have also asked for assistance, and we expect there will be additional requests as well. The Agency plans to provide assistance to all States that request it as rapidly as our available resources and seasonal constraints allow.

The surveys conducted in support of State initiatives primarily focus on locating areas with unusally elevated radon levels. Although this is a different objective than that of our proposed national survey, we are taking care to help the States design their surveys so that the data we derive from them can complement the information obtained from the national survey.

Once States have conducted their surveys, they will be faced with a number of additional tasks to respond to the needs of their citizens. Based on the experiences of other States, such as Pennsylvania and New Jersey, EPA has developed a model of what it considers to be a typical workload for a State which discovers a radon problem within its boundaries. Typical tasks include:

- Development of State measurement capabilities
- Development of State radon diagnostician training
- Development of State contractor training program
- Development of State certification programs
- Development of Home Evaluation Program
- Development of building codes
- Further assessment of geological risk areas
- Development of State risk maps
- Joint demonstration programs (where appropriate)
- Implementation of State loan program (where appropriate)

The Agency, through its Radon Action Program, is also assisting States in developing the necessary capabilities to respond to a radon problem if one is discovered through their survey efforts.

Question 12: What is the nature of the threat to public health posed by naturally occurring radioactivity, including radon, in drinking water? Is it difficult to remedy this problem? When will final regulation on this problem be published?

Answer: The threat to the public health posed by naturally occurring radioactivity in drinking water is primarily due to the presence of three elements, radon, radium, and uranium, in public water supplies. The existing regulations for drinking water cover only radium; radon and uranium were excluded owing to uncertainties in occurrence, exposure routes, and toxicity.

Radon: Radon is found mainly in ground water supplies. While there are several naturally occurring isotopes of radon, only radon-222, the decay product of radium-226, has a long enough half-life to survive transit in a water distribution system. Radon directly ingested in drinking water is less of a threat than the radon liberated from water in other domestic activities, due to lower toxicity, the lesser quantities involved and to disposition in the body. Since radon is an inert gas, it is easily released into the indoor air by showers, baths, washing machines, toilets and dishwashers. The major resulting health hazard is the induction of lung cancer by the radon progeny. The distribution of radon in public water supplies has been measured to some extent. In general, the drinking water contribution to indoor radon is expected to be moderate, (1% to 7% of that from soil) although there are geographical areas where the water supply component would be dominant. Radon is easily removed from drinking water by aeration or by absorption on granular activated carbon.

Radium: Radium is found primarily in underground water supplies. There are two commonly occurring isotopes, radium-226 and radium-228. The risks presented by ingestion of either isotope are roughly comparable with respect to the major health hazard, the induction of bone cancer. Because radium is efficiently removed by standard water treatment methods, it is usually a problem of small supply systems having only limited treatment facilities. The current EPA Maximum Contaminant Level is estimated to be exceeded in about 500 water supplies for each isotope.

Uranium: Uranium is found in both surface and ground waters. It may occur naturally or as a result of human activities. While several isotopes are found in nature, the radiocarcinogenicity of natural uranium must be inferred since its chemical toxicity (acting mainly on the kidney) is predominant. Uranium is not as easily removed from water by standard treatment methods as radium and its removal will probably require additional facilities. The distribution of uranium in public water supplies is not well known but, based on reported data, it is estimated that approximately 1,000 supplies may have levels requiring remedial action.

Work on the final National Primary Drinking Water Regulations has been in progress for several years. It is expected that the regulation will cover both radium isotopes, natural uranium, and radon-222. The Advanced Notice of Proposed Rulemaking has been published (FR, v.51, No. 189, pp. 34836-34862) and the target date for proposal of the regulations is March 1988.

○